New Orleans Medical
and
Surgical Journal

$4.00 Per Annum, 35c Per Copy
Vol. 102, No. 11

MAY, 1950

Published Monthly
1430 Tulane Avenue, New Orleans 12, La.

THE USE OF INTRAVENOUS PROCAINE IN THE TREATMENT OF ARTHRITIS

JAMES R. GREEN, M. D.*
A. A. DOERNER, MD**
AND
E. M. GORDON, M. D.***

The intravenous use of procaine hydrochloride has become more widely accepted as a therapeutic method in recent years than ever before, and has been used in a wide variety of circumstances, such as an analgesic in obstetrics, for control of postoperative pain and dressing of burns in surgery, treatment of serum sickness, cardiac arrythmias, asthma, and the control of pain in the various arthritides. It is the purpose of this report to deal principally with our experience in the use of intravenous procaine hydrochloride in the treatment of arthritis as seen in a general hospital and outpatient practice.

Procaine hydrochloride is an alkaloid, para-amino - benzoyl - diethylaminoethanol, first synthesized by Einhorn over forty years ago in the search for a substitute local anesthetic agent to replace cocaine which has a high degree of toxicity and strong habituation tendencies. Procaine acts quite similarly to cocaine as an anesthetic agent but does not have the severe central toxic action, is nonhabit-forming, is noncumulative, and produces no liver damage; the latter observations have been studied and confirmed by Jacoby, *et al*[1] in their recent reports of observations on the repeated massive but sublethal intravenous administration of novocain to rats and dogs; and in the continuous infusion of 1-2 grams of novocain over a period of one to two hours in humans. They conclude that the intravenous administration of moderate doses of novocain is a safe procedure as regards the effect on the normal liver, as measured by liver function tests.

Because of the recent interest in the use of intravenous procaine for such varied disease states aforementioned, more detailed study regarding the pharmacology of this drug has been carried out. In the devising of chemical methods for identification of procaine and the products of its hydrolysis, Brodie and associates[2] discovered that an enzyme present in plasma was largely responsible for almost instantaneous destruction of procaine to the metabolites, di-ethyl-amino-ethanol and para-amino-benzoic acid; further that 70-95 per cent of the latter drug is recovered unaltered in the urine; whereas amounts of di-ethyl-amino-ethanol equivalent to only 20-35 per cent of the administered procaine can be isolated from the urine. It was also noted that concentration of procaine in the plasma does not increase with continuous intravenous administration while the concentration of di-ethyl-amino-ethanol does increase, suggesting that the latter drug may be the active agent rather than procaine per se. Recently this has been borne out by Rovenstine and

From the Medical Service, U. S. Marine Hospital, New Orleans, Louisiana.

*Resident in Medicine, U. S. Marine Hospital, New Orleans, Louisiana.

**Chief of Medical Service, U. S. Marine Hospital, New Orleans, Louisiana.

***Clinical Director, U. S. Marine Hospital, New Orleans, Louisiana.

Papper[3] in the administration of di-ethyl-amino-ethanol in which they formed the impression that this drug in relatively large doses is equally efficient as procaine as an analgesic.

Besides the hydrolysis of procaine in the blood serum, it is also believed that the liver is instrumental in the breakdown of procaine to its metabolites as well.

It has been observed by pharmacologists in the past that drugs, such as the salicylates, will be found in higher concentration in inflamed tissues. This phenomenon is due to increased capillary permeability which occurs locally in the tissues involved in trauma or inflammatory processes. This concentration in inflamed tissues occurs as well with procaine hydrochloride. It has been shown that procaine concentrates eight times stronger in these inflamed tissues than in normal tissues. The most probable sites of action of procaine and its metabolites in these areas are at the terminal nerve endings themselves, directly, or perhaps by antagonistic action to acetylcholine at myoneural junctions, and by direct action on the endothelium of the blood vessels within the injured capillary bed, or perhaps the antagonistic action against acetylcholine within associated sympathetic ganglia which brings about vasodilatation. An antihistaminic effect has also been noted in treating penicillin sensitivity reactions, serum sickness, and other allergic manifestations with procaine. This latter effect may be partly responsible for the easier breathing of asthmatics, noted incidental to treatment for arthritis. With this in mind we tried procaine, in the same dosage we use for arthritis, on two cases of status asthmaticus who did not respond to the usual drugs. Immediate, although transitory relief was obtained for an hour or two in both cases.

Following the dosage formula as recommended by Graubard and his associates,[4] it was decided to use a 0.1 per cent procaine hydrochloride solution and give 4 mg. per kilogram of body weight and infuse that dose intravenously at a rate not to exceed 14 cc. per minute. One gram of procaine was first dissolved in a small quantity of normal saline and added to 1000 cc. of normal saline. Vitamin C (1 gram per 1000 cc. solution) was also added since it has been demonstrated that many arthritics are low in Vitamin C, and that Vitamin C deficiency leads to a greater incidence of unpleasant side effects during administration of procaine. It is suggested to use 5 per cent glucose in distilled water instead of normal saline as the medium in which to mix the procaine if the patient is a cardiac or on a salt restricted diet for any other reason.

A wheel cart was devised, which could be rolled from one room to another, containing all the necessary solutions, drugs and apparatus in one unit to facilitate administration of intravenous procaine. Although no untoward reactions were encountered in this series of cases, it was deemed advisable to have on hand a soluble barbiturate in the event of any convulsive signs, and metrazol and oxygen for any respiratory distress while procaine was being administered. Aromatic spirits of ammonia were used to counteract occasional vertigo experienced by a few patients.

Although the majority of our patients were hospital cases, they were treated with procaine as an outpatient procedure; that is, they would go to the treatment room, receive procaine and then proceed back to their ward unaccompanied. Outpatients can proceed home within a few minutes after the treatment.

Before starting a new patient on treatment, inquiry was made relative to previous injections with procaine and whether any untoward effects were experienced; since some people are known to exhibit an idiosyncrasy to procaine. With a patient in whom no history can be obtained of taking procaine previously, it would be advisable to skin test with 0.1 cc. of a 1 per cent solution of procaine, and if no systemic signs of toxicity are manifest it is safe to proceed. On the first infusion it is a good practice to run the solution slowly and observe the patient closely.

The first sensation experienced by the

patient during the infusion is a sense of warmth, usually in the epigastric region, about the face, or generalized, and this usually lasts for a few minutes following cessation of a treatment. Some patients experience slight vertigo, which if it becomes marked, can be easily controlled by slowing the rate of infusion. No severe side reactions of note have yet been observed.

A few patients experienced relief of pain during or following the first treatment, but most of them took three infusions over a period of one and one-half weeks before decided improvement was noted. It would seem that before the effect of the treatment in an individual can be determined, he should receive at least three infusions.

Fifty-six cases of arthritis and allied conditions were studied in this series. A total of 220 procaine infusions were given this group.

Twenty-nine patients with osteoarthritis were treated, of whom 21 received marked relief from pain and stiffness; the remaining 8 had transitory relief. In this group labeled osteoarthritis were 3 advanced cases of the Marie-Strümpell type. Two of these patients experienced much relief.

Case No. 1. White male, 40 years old, veteran of World War II, with history of pain in back since 1942. Since that time he had little relief from pain in spite of treatment with hot baths, a three months' course of gold salts therapy, and a course of x-ray therapy to the spine. X-ray revealed arthritis of the spine of the Marie-Strümpell type. The patient weighed 69 Kg. and received 276 mg. of procaine on December 27, 1948. Two more infusions were given during the following week. He then experienced much relief from pain, was discharged from the hospital, and followed for two months as an outpatient, returning once a week for a procaine infusion. He has not required further treatment to date.

Seventeen patients with rheumatoid arthritis were treated, of whom 9 were greatly benefited; the rest received transitory relief from pain. In this group it was observed that the best results were obtained during acute exacerbations of rheumatoid arthritis, rather than with chronic types.

Case No. 2. White male, 23 years old, veteran of World War II, discharged from Navy with rheumatoid arthritis. He had been treated in a Veterans Hospital for six months following discharge from service. He had returned home and was taking codeine, salicylates, and applying liniments to affected joints. When first seen at the hospital he had pain in the temporomandibular joints, knees, feet (including toes) left hip, shoulders, and wrists. One wrist was reddened and swollen. The patient weighed 50 Kg. and was given 200 mg. procaine on December 16, 1948. Later that day he noticed increased mobility in the temporomandibular joints and left hand. During the following week he received two more infusions, at the end of which time he could walk without pain and eat solid food without pain in his jaws. He was allowed to go home over the Christmas weekend and returned with improvement sustained. He was then discharged from the hospital and followed as an outpatient, returning twice a week for the next two months. Improvement was sustained. Patient obtained a job and at this writing is still actively engaged at work.

One patient with chronic brucellosis and continuous back pain, but no x-ray evidence of bony changes, received complete relief with three infusions.

One patient who had muscle spasm following surgery of the knee experienced transitory relief of spasm and pain for three to four days after each treatment.

Three patients with pain in the extremities, incidental to hemiplegia, obtained marked relief of pain and were more readily ambulated.

In a group consisting of one case of fibrositis, one case of subdeltoid bursitis, and one case of acute sacroiliac sprain, fair results were obtained but there were not sufficient cases of these types studied for a good evaluation of therapy.

Although entirely unrelated to the group above, 2 cases of status asthmaticus were given procaine with marked improvement in respiration for from one to two hours.

CONCLUSION

1. Fifty-six patients with various conditions were treated with intravenous procaine.

2. A total of 220 infusions were given, most patients receiving an average of 3 to 4 treatments.

3. Best results were noted in osteoarthritis and acute rheumatoid arthritis.

4. It was also found to be a useful procedure in sprains, fibrositis, muscle spasm following surgery, and pain incident to hemiplegia.

REFERENCES

1. Jacoby, J. J., Coon, J. M., Wolf, M. P. Salerno, P. R. and Livingstone, H. M.: Effect of procaine on liver function, Anesthesiology, 9:481, (Sept) 1948.
2. Brodie, B. B., Lief, P. A., and Poet, R.: Fate of procaine in man following its intravenous administration, J. Pharmacol. & Exper. Therap., in press.
3. Rovenstine, E. A., Papper, E. M.: The therapeutic role of procaine and its derivatives. Bull. New York Acad-Med. 25:298 (May) 1949.
4. Graubard, David J., Robertazzi, Raphael W., and Peterson, Milton C.: Intravenous procaine: A preliminary report. New York State J. Med. 47:2187 (Oct) 1947.

VIRAL HEPATITIS

REPORT OF TWO CASES TREATED WITH CHLORAMPHENICOL

HULON LOTT, M. D.
RICHARD W. YOUNG, M. D.
AND
JOHN C. STOVALL, M. D.
BATON ROUGE

Reimann[1] stated in a recent review of infectious diseases that viral hepatitis resists specific therapy. Two cases are presented which were treated with chloramphenicol.

CASE REPORTS

Case No. 1. L. L., a white male, aged 42, became suddenly ill on November 11, 1949 with chills, fever, headache, and malaise. He had no other symptoms and had been in good health all his life. He had a hemorrhoidectomy fifteen years ago. For the past seven years he had been employed in an oil refinery. There was no contact with industrial toxins, and he denied indiscreet indulgence in alcohol. There was no history of recent venepuncture or inoculations.

The temperature on the third day was 103.4° F., pulse 92, respirations 18, and the blood pressure 134/82 in both arms. His weight was 192 lbs. The eyes, ears, nose and throat were normal. Auscultation and percussion of the chest revealed no abnormality of the heart or lungs. There was, however, tenderness to fist percussion over the area of liver dulness, which extended to the fifth rib anteriorly. The liver was palpable on deep inspiration 3 centimeters below the costal margin. It was tender and the edge was smooth. The spleen was not felt, and the remainder of the abdomen was soft and tympanitic. No other areas of tenderness were noted, and no masses were palpable. The genitals, rectum, skin, bones, muscles, and joints were normal. There was no superficial lymphadenopathy.

The urine analysis revealed a straw color and acid pH. The specific gravity was 1.018. Albumin and sugar were not detected. There was an occasional leukocyte in the sediment. Hemoglobin measured 16.6 grams. Total R.B.C. were 4,950,000. The W.B.C. were 9,150. There were 46 segmenters, 2 bands, 47 small lymphocytes and 5 eosinophiles. Ova of *Enterobius vermicularis* were found in the feces. The serology was negative. The cephalin flocculation was 3 plus in forty-eight hours and the icterus index was 8 units. A chest roentgenogram was normal.

He was placed on bed rest and a dietary intake of 350 grams of carbohydrate, 175 grams of protein and 130 grams of fat. Chloramphenicol was started orally on the fourth day. Initially he was given 1 gram every hour for three doses followed by ½ gram every three hours for a total of seventy-two hours. High potency multivitamins, infusions fortified with vitamins B and C, and choline 1 gram three times daily were given as supportive measures.

The temperature was normal on the fifth day, and he remained afebrile for five days, when his fever returned and ran a low grade course until the fifty-seventh day of the illness. Serial cephalin flocculation tests were 2 and 3 plus on the eleventh, twenty-first, twenty-ninth, and fifty-first days. On the sixtieth day the test was negative and on the sixty-fifth day the thymol turbidity test was 2 units. He has been well since, and subsequently a cholecystogram and gastrointestinal series were normal.

Case No. 2. L. B., a white male, aged 17, developed pain in the right aspect of the epigastrium on December 13, 1949. He had slight nausea. His symptoms had been present two hours when seen. He stated his pain was intermittent and lasted for about five minutes at a time. He had no other symptoms and his health had been good since childhood. There was no history of excess alcohol, recent venepuncture or inoculations.

The temperature was 98:6° F., the pulse 108 per minute, respirations 32, and the blood pressure was 118/72. He was emotionally upset and feared he had appendicitis. The eyes, ears, nose, and throat were normal. The heart rhythm was regular and the sounds were normal. The lungs were clear bilaterally. There was tenderness on fist percussion over the area of liver dulness superiorly to the fifth anterior rib. The abdomen was soft and tympanitic. The liver was palpated 3 centimeters below the right costal margin on deep inspiration. It was moderately tender and the edge was smooth. No masses or other areas of tenderness were noted. The skin, genitals, rectum, bones, joints, and muscles were normal. There was no palpable lymphadenopathy.

The urine analysis was normal. Cysts of *Endolimax nana* were observed in the feces. The hemoglobin was 13.8 grams. Erythrocytes numbered 4,600,000. Total white count was 4,350. The differential was as follows: juveniles 1, bands 9, segmenters 57, eosinophiles 2, basophiles 1, lymphocytes (small) 29 and lymphocytes (atypical) 1. The STS was negative. The cephalin flocculation

From the Baton Rouge General Hospital, Baton Rouge, La.

on the second day was 2 plus in forty-eight hours.

He was placed on a similar supportive regimen as case No. 1. His temperature rose daily to 99° to 101° F. On the ninth day he was passing clay-colored stools, although he was not visibly jaundiced, and the cephalin flocculation was 3 plus. It was again 3 plus on the eighteenth day. On the twenty-fifth day chloramphenicol was begun 1 gram every hour for three doses and then ½ gram every three hours for seventy-two hours, followed by ¼ gram every three hours for an additional forty-eight hours. During therapy with chloramphenicol he remained afebrile. The cephalin flocculation was 2 plus on the next to the last day of therapy. Fever returned seventy-two hours after the antibiotic was discontinued. On the thirtieth day the thymol turbidity was 15 units. During the fifth week of illness there was fever every two or three days of a degree or less. On the forty-fourth day the thymol turbidity was 4.5 units.

COMMENT

The chronicity of viral hepatitis and the invariably final morbid status of the rare instances of cirrhosis as its sequela are indications for rigid coutrol of all patients with this disease. The diet should be pushed to full limits of tolerance, and the adjuncts, vitamins and lipotropic substances, given. Strict bed rest is indicated as long as symptoms and liver function tests reveal hepatic cell damage. In addition, there is a great need for specific therapy. Chloramphenicol was administered to two patients with viral hepatitis. In both there was a remission of fever during therapy with recurrence of fever after the drug was stopped.

CONCLUSION

Chloramphenicol may be efficacious in the treatment of viral hepatitis. Two cases are presented. Prolonged use of chloramphenicol on well controlled cases of "viral" hepatitis is indicated.

REFERENCE

1. Reimann, H. A.: Infectious diseases. Arch. Int. Med. 85:157 (Jan.) 1950.

UNCOMPLICATED GASTRIC AND DUODENAL PEPTIC ULCER*

G. GORDON McHARDY, M. D.†
AND
DONOVAN C. BROWNE, M. D.†
NEW ORLEANS

Desire and obvious necessity for limitation characterize the title of this presentation. Publications, scientific and otherwise, prevail in the medical and lay literature, yet both the nature of this disease and its successful management remain largely hypothetical. However, the ulcer patient, individualized and competently managed by one understanding the disease, does well in "long term" evaluation and rarely develops a complication. Conversely, the situation is an unhappy one fraught with complication where incompetency characterizes the attending physician. This situation probably sponsored Sara Jordan's[1] statement: "The distressing end results of neglect and of ineffective treatment as well as the disabling and depressing effect of the disease itself challenge our best efforts toward the solution of this problem of peptic ulcer," and justify our somewhat routine review of the subject.

Ivy[2] feels that 10 per cent of our population at some time develop a peptic ulcer and that over a ten year period there are aproximately 1,500,000 ulcer patients in the third decade of life. Jennison[3] encountered 7 per cent duodenal ulcers in 2700 unselected fluoroscopic examinations. A cadaveric incidence of 11.85 per cent is recorded by Robertson and Hargis.[4] Portis and Jaffe[5] report ulcers present in 5.2 per cent of autopsies on white people. In ambulatory private gastrointestinal practice (1000 consecutive cases), we encountered peptic ulcer in 22 per cent of patients. Private hospital

---

*Presented at the Second Annual Scientific Session, Academy of General Practice of Louisiana, May 8, 1949, in New Orleans.

†From the Gastroenterologic Clinic of Touro Infirmary and the Browne-McHardy Medical Group, New Orleans, La.

The expenses of this investigation were defrayed in part by a grant from the Medical Research Fund.

(Touro Infirmary) incidence in the past decade revealed a 37.8 per cent incidence of 5853 gastrointestinal admissions and accounted for 3.7 per cent of all hospital admissions during the same period. In the previous ten year period a 26.5 per cent of 8353 gastrointestinal admissions and 4.6 per cent of all hospital admissions was recorded.

Statistical evaluation substantiates Ivy's[2] approximation of a 10 per cent incidence in the adult white male and justifies an impression of increasing prevalence of peptic ulcer.

ETIOLOGY

The genesis of the disease persists speculative. No single factor satisfactorily explains the development of every case of peptic ulcer. Coalition of hypersecretion (hydrochloric acid), hyperirritability (muscular spasm) and tissue susceptibility to the forementioned two undeniably are essential to the production of an ulcer. The primary motivating etiologic influence remains unknown. The contribution of infection, toxic effects (alcohol, smoking, and drugs), trauma (internal and external), vascular disorders, allergy, histamine (used therapeutically), gastritis, nutrition, endocrine, emotion, and a host of other possibly precipitating influences are repeatedly revived. Sex apparently has an unexplained relationship in that duodenal ulcers are significantly more frequent in males. Age seems irrelevant although the third decade of life is the ulcer period.

Inability to reproduce experimentally a peptic ulcer other than that simulating the postoperative gastrojejunal lesion permits considerable speculative freedom which is academically interesting but rarely of practical application. The experimental production of peptic ulceration by histamine, cinchophen, and mecholyl may materialize into a source for adequate study. How important each etiologic possibility is in the individualized ulcer patient is a decision probably essential to intelligent therapeutic application.

SIZE

Consideration of ulcer size, though of interest, has minimal clinical importance. Gastric ulcers averaging 1.5 centimeters are usually larger than the duodenal lesions which average less than 0.5 centimeters.[6]

In relation to malignancy it has been estimated that while 80 per cent of gastric ulcers are less than 1.8 cm. in size, 71 per cent of gastric carcinomas exceed a 4 cm. diameter.[7] While the exception does not prove the rule it often embarrasses it. We have never granted diagnostic or prognostic significance to ulcer size, although we are influenced prognostically by depth indicating degree of penetration.

SITE

That the majority of gastric ulcers are on the lesser curvature in the vicinity of the angularis and rarely occur on the posterior wall is of interest and importance.

Duodenal ulcers are common to the first 2 centimeters beyond the pyloric ring adjacent to the lesser curvature border. Postbulbar ulcers are now being roentgenologically diagnosed more frequently.[8]

While we tend to doubt benignancy in a posterior wall gastric ulcer and anticipate more probable perforation in anterior wall lesions, beyond this we feel locations, like size, lend little to clinical evaluation in the individual patient.

DIAGNOSIS

Characteristic ulcer distress suggests the presence of peptic ulceration. Confirmatory diagnosis depends entirely on the roentgenologic demonstration of either an ulcer crater or anatomic changes produced in the mucosa about the ulcer and by the contraction of the scar tissue in duodenal ulcers. Brown and Harper[9] stress the importance of the right lateral horizontal roentgen study. Gastroscopy complements roentgenology in the study for gastric ulceration.[11] In meticulous diagnostic study of 200 consecutive cases, in which the clinical evaluation insisted on the diagnosis of peptic ulcer, the roentgen study was confirmatory in 83 per cent; gastroscopy gave a supplemental 2.5 per cent to bring the confirmed incidence to 85.5 per cent. In the elimination of ulcer simulating disease adequate explanation was revealed elsewhere in 7 per cent. Therapeutic response to ulcer management permitted us to haz-

ard a diagnosis of "ulcer occulta" on the remaining 7.5 per cent of cases. Such a diagnosis requires constant observation and repeated diagnostic surveys to substantiate itself and has never given us a sense of security in handling the patient.

Among the diagnostic aids one must mention gastric analysis. Histamine achlorhydria never occurs in patients with an active peptic ulcer. Gross blood in the gastric content suggests an ulcerating lesion. Cytologic and chemical study of the gastric secretion may aid in establishing the existence of neoplasia. Melena is highly significant. The hematologic picture is not conclusive; anemia is expected with chronic blood loss from ulceration; conversely, duodenal ulcer is reported concomitant with polycythemia.

Gastrophotography, Woldman's phenolphthalein test, gastrographic observations, and Einhorn's string test are mentioned only for completeness.

We are not projecting this manuscript beyond benign lesions other than to state that all gastric lesions are suspected of malignancy until their benignancy is assuredly proved.

HEALING

Factors influencing healing include proper advocation of therapy, acceptance of the regime, and actual potentialities of healing. The size of the lesion, the amount of scar tissue adherence of the ulcer base to contiguous structures, and the age of the patient, each is concerned in determining potentiality of healing. Complete healing may never occur in some cases. When the ulcer history indicates long duration with brief remission periods in a patient beyond 45 years of age, complete or rapid healing is not anticipated.

Disappearance of the roentgen niche often occurs early; while prognostically favorable it is not criteria for complete healing.

In general, it is conceded that a gastric ulcer leaves no scar unless the ulcer has been very large and deep. Penetration often results in extensive distortion by scarring in the process of healing. While many anterior wall duodenal ulcers heal with minimal gross scarring most of those located on the posterior wall form extensive cicatrization and concomitant lumen constriction. The postpyloric ulcer often by scarring lumen constriction produces a megaduodenum and the potentiality of prestenotic pseudodiverticulum of the cap area.

Our roentgen follow up of 100 duodenal ulcer cases, asymptomatic for twelve months, revealed a demonstrable crater present in 12 and a scarred or deformed duodenum in 20 additional cases. Templeton[10] remarks: "In most instances a deformity of the bulb changes not at all or only slightly as the crater disappears." Fifty gastric ulcers, of the same period of quiescence, showed no roentgenologic or gastroscopic evidence of the previous site of ulceration. In a study of 200 instances of ulcer recurrence the crater was found at the site of the original lesion in 86 per cent indicating to us the probability that complete healing of the ulcer had not occurred. We have not been able to duplicate the remarkable average time for healing duodenal ulcers (thirty-seven days) approximated by Cummings, Grossman and Ivy.[11]

CURE

We consider as two distinct entities the duodenal ulcer and the gastric ulcer. To evaluate our management of the ulcer patient we completed two ten year follow-up surveys. The first of these, previously reported in part, was completed in 1938, the second in 1948. Meticulous case study and personal interview, in preference to questionnaire inquiry, were elected. Therefore, neither is a series of consecutive cases and each ten year study was limited to 100 duodenal initially uncomplicated ulcer patients. The study included only patients hospitalized for initiation of therapy. This composite study revealed that 98 individuals so managed may be classified as ten year "cures". Thirty-eight were considered satisfactory in that recurrent symptoms were easily managed; they remained uncomplicated, disability was insignificant and more or less an elective state. There were 22 un-

satisfactory results in that, despite lack of demonstrable complication, recurrences, though not severe, were frequent, refractory, and the degree of disability created an economic problem. Six who had surgery against our advice are partially disabled. The remaining 42 developed ulcer complications and from this viewpoint were unsatisfactory. These included 27 instances of hemorrhage; 16 were medically managed through a single bleeding episode, 7 through multiple exsanguinating illnesses; 3 required emergency surgery. Surgical management of acute perforation in 6 instances was successful. Medical management of 2 perforations (surgery refused) resulted in uneventful recovery from acute illness. There were 19 obstructive ulcers; 12 of these were measured to be of beyond 50 per cent six hour retention with lack of therapeutic response, and were therefore advised to have elective surgical correction. Ten consented, 2 have continued on a medical program. The remaining 7 whose retention is less than 50 per cent and intermittent are still under continuous medical observation. There was no instance of coexistent gastric carcinoma with duodenal ulcer in this series.

In summarization, this critical analysis of our own management of duodenal ulcer patients revealed a comparable result in the two ten year periods, indicating no evidence of improvement in our therapeutic program.

98 or 49 per cent are classified as "cures."
38 or 19 per cent are satisfactory results.
22 or 11 per cent are uncomplicated unsatisfactory results.
42 or 21 per cent are complicated poor results.

A clinical and physiological difference exists between gastric and duodenal ulcers, possibly a consequence of location of the lesion and related to inhibitory secretory and motor mechanisms present in the normal duodenum. This reasoning loses value when one observes that a gastric ulcer concomitant with a duodenal ulcer heals more readily than the duodenal lesion.

In the period there were 86 instances of gastric ulcer similarly available for survey. Seventy-four healed and remained healed, 12 recurred and were resected. From this limited study one would gather confirmation that benign gastric ulcers respond to therapy more satisfactorily than do duodenal ulcers. In no instance of this series, nor in 52 additional cases of gastric ulcer with a five year follow-up, have we reversed the initial diagnosis when confirmed by therapeutic response, and we therefore incline toward medical management of gastric ulcers with preservation of gastric physiology despite the surgical concept of resection reassurance against malignancy.

Recurrences in duodenal ulcer by annual estimation revealed 15 per cent in the first year, 21 per cent in the second year, 11 per cent in the third year, 6 per cent in the fourth year, and only 3 per cent in the remaining five years. This would tend to indicate a five year evaluation to be fairly accurate but does not conform to the findings in Flood's[12] excellent statistical evaluation showing recurrences every year through a fifteen year follow-up.

Our statistics, conforming well with those of early and undenied reports of Jordan and Keefer, Crohn, St. John and Flood,[13] do not permit optimism or contentment with our present management of the ulcer patient.

This therapeutic story does not approximate that of those happy authors advocating their new and successful methods of management where a "cure" is reported in an average 87 per cent but it must at once be recognized that we are interpreting our results from accurate ten year follow-ups. We therefore feel that our statistics give a fair evaluation and permit us to claim a studied estimate of the ulcer situation and its control.

TREATMENT

In the management of the peptic ulcer patient we wish to gain three points: (1) symptomatic relief, (2) ulcer healing, and (3) recurrence prevention.

In the sincerity of investigation a complete medical diagnostic survey in a methodical manner is specified. This has a reassuring influence on the skeptical patient and is thereby a part of therapy. It may

necessitate an informal neuropsychiatric survey dwelling on analysis and perhaps adequate endocrine study. A sincere attempt to evaluate allergy and the place of tobacco in those possibly sensitive, an effort to estimate rest requirement, mental and physical, and a decision as to whether ambulatory or hospital medical management is to be elected are each exacted. From this thoroughness and individualization one approaches the treatment of the person with the ulcer as well as institution of measures directed toward the cure of the ulcer itself. We must therefore create for ourselves and in our patient a broad concept of peptic ulcer disease. The physician and patient each reviewing the disease estimate its relationship to various etiologic possibilities and make adequate corrective measures before entering into the routine of caring for the ulcer itself. Then, speaking in terms clearly understood by the patient, a program is agreed to. Several principles are exacted. The gastric ulcer case always is hospitalized for initiating therapy, the gastric ulcer must heal roentgenologically and gastroscopically within eight weeks and stay healed or it becomes a surgical problem. A two year program is anticipated in the cure of both gastric and duodenal ulcer patients. Gastric ulcers are roentgenologically checked every three months after healing for two years and then annually for life. Duodenal ulcer patients are roentgenologically checked semiannually from the onset for two years.

In duodenal ulcer patients the election to hospitalize an individual is often an essential to evaluation. Separated from environmental influences the personality type becomes more obvious, rest becames possible, relaxation practical, dietary and therapeutic measures easily applicable. Tanner,[14] in addition, indicates recumbency to diminish gastric activity, decrease venous stasis, increase blood supply, and favorably alter the gastric acidity. It is obviously true to the observant physician that symptomatic response is more rapid in hospitalized patients; that they are subsequently more easily managed, and we therefore favor such management where practical.

The basic therapy beyond measures to achieve physical and mental rest are all extended to obtain gastric rest whereby hyperirritability and hypersecretion are controlled. These measures considered individually include (1) diet; (2) antacids; (3) constant drip measures; (4) drugs of antispasmodic and sedative action; (5) incidental measures: (a) mechanical, (b) parenteral preparations, (c) gastric radiation, (d) hormones, enterogastrone and mucosal extracts, (e) psychotherapy.

DIET

Diet is probably the most important part of the program. The diet must be practical, calorically adequate, individually applicable, relatively devoid of gastric motor and secretory stimulation. The program should be stipulated as frequent small feedings, whereby quantitative gastric distention with resultant peristaltic contraction is eliminated. Small feedings excite less secretory response; frequency permits better secretory neutralization.

Secretagogues such as meat extracts, seasoning, condiments, and alcohol are eliminated. Cellulose and meat fibers, considered possibly traumatic, are restricted. Semiliquid consistency would indicate minimal requirements for macerating function. Carbohydrate leaves the stomach too rapidly; fats remain in the stomach too long. The ideal therefore seems to be a combined food.

The ideal food is milk. Milk allergies (5 per cent) or idiosyncrasies exist, and when recognized, suitable substitute such as gelatins, cream soups, strained chicken and lamb broth, and strained cooked cereals must be used. Conception of milk sensitivity may be ameliorated by boiled milk, evaporated milks, addition of lime water and non-allergic preparations.

Our concept of the dietary management is a four step program:

1. The hospital ulcer dietary regimen is insisted upon for all gastric ulcer patients and advocated for all duodenal ulcer patients. Only when circumstances arising from hospitalization create too great an or-

deal for the individual do we agree that the program might best be initiated with the patient ambulatory.

2. The ambulatory ulcer dietary regimen should closely adhere to the program initiated in the hospital with hourly feedings, as charted, when possible, until the physician is convinced that healing has progressed satisfactorily, and then is changed at an arbitrary six weeks to the early ulcer diet.

3. When a three month satisfactory symptomatic response has been achieved we then advocate a normal ulcer diet, which is adhered to for approximately eighteen months. The patient is advised to revert back to his earliest program upon the slightest recurrence of symptoms, during acute respiratory and other infectious illness, when under unusual mental or physical stress and at the time of anticipated seasonal activation. Timing of feeding may be, of course, adjusted to the patient's working hours but we do not feel that ulcer patients do well on alternate shifts as exacted in some industries.

Susceptible to exploitation, the ulcer diet has been often dramatically supplemented by nutritional adjuncts. Protein hydrolysates are accredited with creditable buffering action and a valuable source of nitrogen intake; there is, however, no conclusive evidence that they are therapeutically indicated since a protein deficiency is not uniformly present in ulcer patients, nor are they as efficient as standard antacids. Ascorbic acid should be added to or included in the diets of patients in all instances, since there is convincing evidence in the literature indicating an actual need.

ANTACIDS

Antacids are almost unanimously endorsed as essential therapy. Complete neutralization of gastric acid-pepsin secretion may be possible but hardly practical other than for hospitalization periods. It is not considered essential to satisfactory ulcer management. However, reduction in acid-pepsin gastric content is conducive to control of ulcer manifestations in all instances, and especially when hypersecretion and hyperacidity are prominent.

Seniority and common usage keep ever prominent the soluble antacids,—sodium bicarbonate, magnesium oxide, and magnesium carbonate. Less frequently used alkalis, such as calcium carbonate, sodium and potassium citrates, tribasic calcium and magnesium phosphates, and the bismuth salts, are often used in various patented alkaline powders. A combination of calcium carbonate with small amounts of magnesium oxide and one of the bismuth salts is the most efficient combination with the least deleterious effect. Bismuth subcarbonate is applicable in the presence of diarrhea. While both efficient, pleasant, and inexpensive, it must be remembered that in using soluble alkali freely one risks the danger of change in acid-base balance of the blood secondary hypersecretion, and renal calculi. In powder or tablet combination they are still widely used and when properly applied deserve their prominent therapeutic position.

Nonabsorbable antacids, such as colloidal aluminum hydroxide and phosphate, both in the reactive and non-reactive forms, have gained overwhelming favor in recent years. We avoid their use in stasis since they have a tendency to delay gastric emptying. The formation of aluminum chloride, irritating to the stomach unless alkali is present to reconvert it, is a disadvantage. In deficiency of pancreatic juice or in diarrhea they may produce phosphorus deficiency. These side effects, plus anorexia with resultant nutritional disturbance and constipation, detract from their efficiency.

Magnesium trisilicate in combination with colloidal aluminum hydroxide is more effective than aluminum hydroxide alone. It tends to combat the costive action of aluminum hydroxide, but alone lacks the neutralization efficiency and astringency of aluminum hydroxide.

Resins,[13-15] reputed symptomatically effective but lacking ability to prevent complications, are expensive. They have negligible toxicity, and minimal side effects of unpleasant taste, nauseating and emetic in-

fluence. In our experience resins in their limited efficiency have their only advantage in that the patient is offered something different.

Protein hydrolysates [16-18] are nutritional adjuncts and not efficient acid-pepsin buffers. Despite enthusiastic reports, there is no conculsive evidence that hydrolysates are superior to a standard antacid regimen, and they are both expensive and distasteful.

Gastric mucin,[21] vegetable mucin,[22] and pectin[23] apparently have too little neutralizing power, are difficult to administer, expensive, and seem inadequately evaluated for the endorsement given them by the few reporting favorable response.

The antacid chosen must be given frequently, hourly when hospitalized, and where practical, in the initial ambulatory period. Tablet form is convenient for the patient but must be given in adequate quantity.

CONSTANT DRIP THERAPY

When neutralization is unsatisfactory, or symptomatic response is lacking in the patient with an uncomplicated ulcer, whose dietary outline is proper, and on whom the use of neutralizing agents has been intelligently applied, there may exist an indication for constant drip therapy. We frankly feel this is best applicable for its psychic influence on the disgruntled, refractory patient for whom something different must be offered. This is the only indication we appreciate for its application, and we only favor the use of the aluminum hydroxide drip.

The intragastric alkalinized milk drip of Winkelstein introduced in 1933, and the aluminum hydroxide drip endorsed by Woldman in 1936, each has a rational basis. Soresi's continuous nutrient drip into the esophagus is unphysiological and irritating. Young's penicillin intragastric drip lacks a rational basis and confirmation. Jejunal alimentation advocated by Einhorn is not applicable to the uncomplicated ulcer case.

DRUGS

Drugs other than antacids in ulcer management are few. To influence motor and secretory activity one of the most commonly used drugs is an antispasmodic or supposed antispasmodic. Should spasm exist and be symptomatic, which is more often presumed than actual, an effective antispasmodic would be a therapeutic triumph, but none effective and long acting, free of distressing side effects, is available. It is true belladonna and its alkaloid atropine depress or abolish motility but they have no demonstrable effect on tonus. The synthetic antispasmodics have not lived up to their reputed efficiency.[19, 20] Papaverine, because of its habit-forming potentialities, has limited applicability. Tetraethyl-ammonium chloride has been reported to be an effective pain reliever with minimal side effects, no effect on motility, and possibly some secretory inhibition, theoretically acting as a circulatory alternant, but it must be administered parenterally and is only considered for hospitalized patients.

Despite lack of proved value we favor belladonna in the presence of persistent pain and in instances of pyloric obstruction. We cannot physiologically rationalize a claim that a medical vagotomy is achievable by atropinism.

Sedatives aid in achieving rest, and in allaying anxiety and emotional tension. As such they are also antispasmodics, and certainly enhance the effectiveness of the presumed antispasmodics. Preference for barbiturate over bromide is only with reference to ease of administration; idiosyncrasies to various sedatives are individual problems.

INCIDENTAL MEASURES

The continuous or intermittent mechanical removal of gastric content in stasis and hypersecretion, both therapeutically and in evaluation of therapy, has definite applicability especially in instances of retention.

Nonspecific proteins and vaccines, among the more prominent of which are milk protein (Aolan),[24] lipoproteins with emetine[25] (Synodal), polyvalent respiratory vaccine[26] along with histadine (Larostidin)[27] and histamine desensitization;[28] each lack rationalization, and efficacy is discounted in control studies.

Radiation therapy, by Palmer and his associates,[29] producing a period of achlorhydria adequate to permit ulcer healing, is apparently effective in proportion to the reduction in gastric secretion. Evaluation by these competent observers is impressive, but the incidence of recurrence admitted detracts from the enthusiastic therapeutic claims. Hamilton[30] has reported assumed gastric ulcer production following radiation.

The hormonal approach, still under experimental study, gives hope for ulcer recurrence prevention either by reduction in acid secretion or by increasing mucosal resistance. Gastric, duodenal, and intestinal mucosal and submucosal extracts are under consideration. Entero-gastrone,[31-33] widely discussed now, appears an impractical, expensive, inadequate measure in well controlled studies recently completed. Rivers[34] and others have hope for the gastric and duodenal extracts, but their actual status is as yet undetermined. Extract of pregnant mare's urine, in the opinion of Page,[35] deserves continued evaluation.

Interest in psychosomatic medicine is so intense, and stress and strain, anxiety and uncertainty, are so prevalent in our present era that there is a tendency toward formal psychiatric management in the ulcer patient. Zane[36] claims "tension" a common factor in all cases psychiatrically studied. Moses[37] studied ulcer cases characterized by insecurity with electroencephalography and classified them dominant alpha index. It is admitted that of the somatic diseases influenced by the psyche, that is, diseases other than the neoplastic and infectious, the psyche enjoys its greatest character role in peptic ulcer. Nevertheless, the somatic element is dominant with variation in relative psychosomatic over somatopsychic interplay. This involves only good application of the "art of medicine" in the individualization management of the ulcer patient with formal psychotherapy applicable only when the separate psyche entity is a departure from the ulcer problem.

The psychosomatists have attempted a division into four different types of ulcer personality:

1. Aggressive, ambitious, successful, independent.
2. Energetic, obsessive, shy, successful, dependent.
3. Shy, not aggressive, unsuccessful, completely dependent.
4. Character disturbances, irresponsible, psychopathic.

It would conceivably be possible to classify our patients with major diseases in the same fashion. It is difficult to discern what practical application such has to the ulcer problem.

New methods and new drugs for the treatment of peptic ulcer appear with monotonous frequency. A complete list of various measures advocated and discarded as a part of ulcer management would approach indexing proportions and would only serve to indicate conservatism and justifiable skepticism in accepting "short term," uncomfirmed therapeutic measures. In this respect it is interesting to review the recent evaluation of various therapeutic possibilities on Mann-Williamson dogs by Saltzstein and his associates.[38]

RECURRENCE PREVENTION

From our survey the conclusion is obvious that we can hope for "cure" in only approximately 50 per cent of duodenal ulcer cases. In an effort to determine the precipitant of recurrent symptoms we evaluated the factors concerned as ascertainable in 500 cases with a recurrent history. No satisfactory explanation for reactivation could be elicited in 27 per cent. Dietary indiscretion, including injudicious use of alcohol, was the most frequent source of recurrence, representing 31 per cent. Emotional trauma, prominent in the form of insecurity, was present in 16 per cent. Unusual fatigue accounted for 10 per cent. A seasonal influence was suggested in 8 per cent. Smoking was a possibility in 5 per cent. Infection of significant relationship was present in 2 per cent. External trauma was suggested in 1 per cent. However, in 62 per cent of these same cases with multiple recurrences, a different precipitant was accused for another episode of ulcer symptomatology. This leaves incon-

clusive assistance to any plan of therapy for recurrence prevention other than continued careful ulcer patient management.

CONCLUSION

The ulcer patient presents an incompletely solved problem of increasing magnitude. With etiology uncertain, it follows that therapy must be speculative to a considerable degree. There is evidence of general admission that the true nature of the disease anticipates a majority of "cures' 'for gastric ulcer, a reasonable percentage (50 per cent) of "cures" for duodenal ulcers. Since this treatise deals with uncomplicated ulcers, there is no comparative therapeutic issue with surgical measures. There is, in our opinion, justification for reservation in the enthusiastic attitude of the surgeon for attacking all gastric ulcers. In general, we feel the uncomplicated ulcer patient has every reason to be satisfied with his prognosis even though the physician can hardly be content and should continue search for the unknown that will increase tissue resistance since he cannot permanently control gastric hypersecretion and hypertonicity. Nevertheless, we should not endorse, without evaluation, the constant influx of therapeutic panaceas thrust upon the ulcer patient.

Long term evaluation with consideration of the ulcer patient like the diabetic, "once a priest always a priest" inculcation of this concept in the patient and physican probably promises the best result in our present therapeutic limitation.

Note: Since this article was submitted for publication, we have treated a controlled group of thirty-dine duodenal ulcer patients with Banthine (SC 1703) (G. D. Searle and Co.) with complete symptomatic relief but no roentgenologic evidence of satisfactory ulcer healing in a 120 day follow-up.

REFERENCES

1. Jordan, Sara M.: Peptic ulcer: A diagnostic and therapeutic problem, Gastroenterology 9:237, 1947.
2. Ivy, A. C.: The problem of peptic ulcer, J. A. M. A. 132:1053, 1946.
3. Jennison, J.: Observations made on a group of employees with duodenal ulcer, Am. J. M. Sci. 196:654, 1938.
4. Robertson, H. E. and Hargis, E. H.: Duodenal ulcer, M. Clin. North America 8:1065, 1925.
5. Portis, S. A. and Jaffe, R. H.: A study of peptic ulcer based on necropsy records, J. A. M. A. 110:6, 1938.
6. Bockus, H.: Gastroenterology, Vol. 1, p. 355, 1943, W. B. Saunders Co.
7. Alvarez, W. C. and McCarty, W. C.: Sizes of resected gastric ulcers and gastric carcinomas, J. A. M. A. 91.226, 1928.
8. Falla Alvarez, L. and Farinnas, P. L.: Postbulbar duodenal ulcers, Gastroenterology 8:1, 1947.
9. Brown, S. and Harper, F. G.: Roentgen diagnosis of duodenal ulcer in the right lateral decubitus position, Rodiology 47:575, 1946.
10. Templeton, F. E.: The crater in uncomplicated duodenal ulcer. Its significance in diagnosis and treatment, Am. J. Roentgen. 59:87. 1948.
11. Cummings, G. M., Jr., Grossman, M. L. and Ivy, A. C.: A study of the time of "healing" of peptic ulcer in a series of sixty-nine cases of duodenal and gastric craters, Gastroenterology 7:20, 1946.
12. Flood, C. A.: Recurrence in duodenal ulcer under medical management, Gastroenterology 10:184, 1948.
13. (a) Jordan, S. M. and Keeffer, E. D.: Factors influencing prognosis in the medical treatment of duodenal ulcer, Am. J. Surg. 15:472, 1932.
(b) Crohn, B. B.: Recurrences in Peptic Ulcer, Lahey Birthday Volume. June 1940.
(c) St. John, F. B. and Flood, C. A.: A study of the results of medical treatment of duodenal ulcer. Ann. Surg. 110:37, 1939.
14. Tanner, N. C.: The management of peptic ulcer. Bristol Med. Chir. J. 63:16, 1946.
15. (a) Segal, H. L., Hodge, H. C., Watson, J. S. and Coates, H.: A Polyamine formaldehyde resin, Gastroenterology, 8:199, 1947.
(b) Spears, M. M. and Pfeiffer, M. C. J.: Anion exchange resin and peptic ulcer pain, Gastroenterology, 8:191, 1947.
(c) Kraemer, M. and Lehman, D. J., Jr.: The treatment of peptic ulcer with anion exchange resins, Gastroenterology, 8:202, 1947.
16. Woldman, E. E., Fishman, D., Knowlton, R. S., Rousuck, A. A., Stover, W. C.: Evaluation of protein hydrolysate therapy for peptic ulcer, J. A. M. A. 137:1289, 1948.
17. Hodges, H. H.: Protein hydrolysate therapy for peptic ulcer, Gastroenterology, 8:476, 1947.
18. Kenamore, B., Lonagan, W., and Shy, J. D.: Protein hydrolysate therapy in peptic ulcer, Gastroenterology, 10:117, 1948.
19. Alvarez, Walter: Are antispasmodics of much value? Gastroenterology 12:155, 1949.
20. Lorber, S. H. and Machella, T. E.: The effect of syntropan on the motor activities of the human gastrointestinal tract and on gastric acidity, Gastroenterology, 12:57, 1949.
21. Fogelson, S. J.: Gastric mucin treatment for peptic ulcer , Arch. Int. Med. 55:7, 1935.
22. Meyer, J., Seidmon, E. E. and Necheles, H.: The treatment of peptic ulcer with powdered okra, Illinois M. J. 64:339, 1933.
23. Winters, E. E., Peters, G. A. and Crook, G. W.: Pectin as a prophylaxis and curative agent for peptic ulcers produced experimentally with cinchophen, Am. J. Digest. Dis. 6:12, 1939.
24. Martin, L.: Peptic ulcer, Arch. Int. Med. 43:299, 1929.
25. Cunha, F.: Experiences with new mode of treating peptic ulcer, Am. J. Surg., 23:219, 1934.
26. Sandweiss, D. J. and Meyers, S. G.: Treatment of peptic ulcer with bacterial vaccines, Am. J. Digest. Dis. 1:338, 1934.
27. Sandweiss, D. J.: Treatment of gastroduodenal ulcer with histidine monohydrochloride, J. A. M. A. 106:1452, 1936.
28. Bernstein, B. M.: Histamine in the treatment of peptic ulcer, Ann. Int. Med. 26:852, 1947.
29. (a) Palmer, W. L. and Templeton, F.: The effect of radiation therapy on gastric secretion, J. A. M. A. 112:1429, 1939.
(b) Ricketts, W. E., Palmer, W. L. and Hamann, A.: Radiation therapy in peptic ulcer, Gastroenterology, 11:789, 1948.
(c) Ricketts, W. E., Palmer, W. L. and Hamann, A.: Radiation therapy in peptic ulcer. A study of selected cases, Gastroenterology, 11:807, 1948.

(d) Ricketts, W. E., Kersner, J. B., Humphreys, E. M. and Palmer, W. L.: Effect of roentgen irradiation of the gastric mucosa, Gastroenterology, 11:818, 1948.

30. Hamilton, F. E.: Gastric ulcer following radiation, Arch. Surg. 55:394, 1947.

31. (a) Grossman, M. I.: An attempt to prevent histamine-induced ulcers in dogs by the administration of enterogastrome concentrates, Gastroenterology, 6:145, 1946.

(b) Grossman, M. I., Greengard, H., Dutton, D. F., Wooley, J. R.: The effects of prolonged administration of enterogastrone on gastric secretion in normal and Mann-Williamson dogs, Gastroenterology, 2:437, 1944.

(c) Greengard, H. Atkinson, A. J. Grossman, M. I. and Ivy, A. C.: The effectiveness of parenterally administered "enterogastrone" in the prophylaxis of experimental and clinical peptic ulcer, Gastroenterology 7:625, 1946.

32. Levin, E., Kirsner, J. B. and Palmer, W. L.: Preliminary observations on histamine and insulin stimulated gastric secretion during the injection of an enterogastrone concentrate in man, Gastroenterology, 10:274, 1948.

33. Ferayorne, R. R., Colle, C. F. and Morlock, C. G.: The effect of enterogastrone concentrates on gastric secretion in human beings, Gastroenterology, 11:730, 1948.

34. Rivers, A. B.: Extract of duodenal mucosa and submucosa in the treatment of peptic ulcer, Proc. Staff Meet., Mayo Clin. 9:665, 1934.

35. Page, R. C. and Heffner, R. D.: Oral treatment of chronic duodenal and jejunal ulcers with an extract of pregnant mare's urine, Gastroenterology, 11:842, 1948.

36. Zane, M. D.: Psychosomatic considerations in peptic ulcer, Psychosom. Med. 9:372, 1947.

37. Moses, L.: Psychodynamic and electroencephalographic factors in duodenal ulcer, Psychosom. Med. 8:405, 1946.

38. Saltzstein, H. D., Sandweiss, D. J. and Hill, E. J.: Results in treatment of 374 Mann-Williamson dogs, Gastroenterology, 12:122, 1949.

DISCUSSION

1. Question: What is the rationale of vitamins, especialy parenterally, in ulcer therapy?

Answer: In the first place, there is no indication for vitamins in any disease unless there is clinical evidence of deficiency. It is commonly felt that the average diet given an ulcer patient is deficient in vitamin C. The diet we outline omits the citrus fruit juices and other direct sources of vitamin C and we therefore supplement it with vitamin C. If prolonged illness in ulcer patients has produced deficiency, vitamins are given where indicated. Besides Vitamin C, a frequently indicated one is B complex, theoretically because of a hepatic deficiency. As common practice we rarely find indication for vitamins other than vitamin C as a supplement to diet. As to giving vitamins parenterally I fail to see any reason unless it be, (1) that the patient is unable to swallow vitamins because of esophageal obstruction; (2) retention in the stomach; or (3) that you feel there is adequate proof that the patient has some disease that will prevent absorbing vitamins; or (4), if you want to use the administration of vitamins to keep your patient coming back. I am not saying that maliciously—sometimes you have to have a reason to get the patient back at intervals and if you want to use vitamins to obtain the patient's cooperation that may be justified. That is my attitude on vitamins.

2. Question: What is your opinion of the use of amino acid and protein hydrolysates?

Answer: Dr. Jerome Levy in Little Rock is credited with being the first man in this country fostering the use of protein hydrolysates in ulcer patients. Then Co-Tui who made Reader's Digest, claimed such remarkable efficiency for protein hydrolysates. Co-Tui had to retract some of his statements. A number of papers have appeared in the literature and there is a great deal of conflict in conclusions reached. Some feel that protein hydrolysates do have buffering action on gastric acidity; others emphatically disprove this.

Protein hydrolysates are a nutritional adjunct. We use these orally in limited amount, because they are expensive and nauseating.

When patients are in the hospital and are being prepared for surgery or have complete lack of nourishment by mouth as part of ulcer therapy we do give intravenous protein hydrolysates cautiously because most of the products available result in unpleasant reactions. We have found Parenamine (Winthrop) the most efficient preparation. It can be given in a reasonable period of time without reaction. We favor it to replace infusions as a part of ulcer management.

We have found that the reactions to intravenous amino acid can be ameliorated or prevented entirely by using benadryl before infusions or along with them.

Dr. D. N. Silverman (New Orleans): I think our knowledge of cause and treatment of peptic ulcer may go back to the very thorough study of pathology of the disease by Bress. We can understand from his studies why it is so difficult to treat an ulcer which is a lesion that has at the same time such chronic inflammatory degenerative and proliferative pathology. All too often we have thought we have cured such an ulcer and have found at operation and autopsy most of these lesions, in a stage of remission and apparently cured by radiologic examination, were still active ulcers. In newer observations I have found that it is most surprising that there are so many so-called silent ulcers. These observations have come about as a result of the study of our dysenteries, amebic and bacillary. Such cases did not yield completely to treatment in the sense of receiving remissions of symptoms or cure of the disease apparent. On further investigation of several such cases of amebic and bacillary dysentery we found the reason for failure of cure was presence of so-called peptic ulcer, silent as far as symptoms were concerned before and during the treatment for the dysentery.

With reference to treatment for dysentery I think as Dr. McHardy and Dr. Brown have brought out we have to be very judicious and careful about use of drugs. That applies to new drugs in the treatment of gastrointestinal disease. We have observed in several patients examined previously without evidence of ulcer, radiologically, after administration of certain antiamebics or

amebicides, these patients presented upper abdominal symptoms ordinarily attributed to ulcer and showed on radiologic examination gastric ulcer which they did not present before dysentery.

CONGENITAL MALROTATION OF THE MIDGUT IN INFANTS

REPORT OF FOUR CASES

BROOX C. GARRETT, M. D.
AND
L. J. O'NEIL, M. D.
NEW ORLEANS

Malrotation of the midgut of the intestinal tract in infants is amenable to surgery. The condition, however, is relatively rare. About 225 cases have been reported in the world literature, and Cohen states that in 6,000 births at the New York Jewish Maternity Center there were only 6 cases.

A review of the development of the gastrointestinal tract in the embryo discloses how any defect in the normal pattern could produce symptoms and even death if proper surgical therapy is not instituted quickly and adequately.

EMBRYOLOGY

From the sixth to the tenth week of embryonic life the alimentary tube grows at a faster rate than the celomic cavity. The growth of the liver and midgut soon fills the intra-abdominal space and the midgut is forced into the root of the umbilical cord as a temporary umbilical hernia. At the tenth or eleventh week, the peritoneal cavity grows at a greater rate than the gut and the midgut is withdrawn into the abdomen. As the return begins the proximal coils of the small intestines are reduced first, and behind the superior mesenteric artery, while the cecum returns last, and it and the transverse colon come to lie in front of the mesenteric vessels. As the midgut recedes into the abdomen it rotates in a counterclockwise manner; a 270 degree counterclockwise rotation takes place. The terminal ileum, cecum, ascending colon, and proximal half of the transverse colon at first lie wholly in the left side of the abdomen, but after this rotation takes place they come to lie in their normal positions. The cecum and ascending colon develop their peritoneal attachments and reflections to the right abdominal wall and the mesentary of the small gut becomes attached to the posterior abdominal wall, obliquely downward from the left upper quadrant to the right lower quadrant. Thus the normal rotation of 270 degrees is completed.

Presented at meeting of the Orleans Parish Medical Society, October 10, 1949.

ETIOLOGY

Any defect in this rotation will give rise to abnormalities which are characterized by (1) an incompletely rotated cecum, (2) a lack of attachment of the mesentery along the posterior abdominal wall, or (3) a completely rotated cecum which is mobile and unattached.

If the cecum has not completed its rotation it is usually found in the right upper quadrant with bands of adhesions reflected from it to the right posterior abdominal wall, across the descending duodenum. These may be taut enough to give symptoms of high intestinal obstruction. If the cecum has proceeded slightly farther in its rotation it may lie directly on the duodenum and cause symptoms by direct pressure.

Associated with this malposition of the cecum is the fact that the mesentery of the small bowel does not have its usual fixation to the posterior abdominal wall, but has only a very short rudimentary attachment just below the origin of the superior mesenteric artery. The entire midgut may twist around this stalk and a volvulus of the entire midgut results. When this occurs it is usually in a clockwise fashion and may be from one to four complete turns. This condition, when it exists, is most dangerous and urgent, as not only obstruction has to be dealt with but infarction of the entire midgut may occur.

DIAGNOSTIC POINTS

Over half of the cases of malrotation produce symptoms before the third week of life; however, the condition may remain silent for some time and produce symptoms later in childhood. These symptoms are much like those of coeliac disease, and a barium enema is necessary for diagnosis.

The clinical picture usually seen with

duodenal obstruction from congenital bands is persistent vomiting, and the vomitus contains bile. Abdominal distention is present and in the early phases is limited to the epigastrium, but later may be generalized due to fermentation beyond the obstructed duodenum. The child may have scanty stools as the obstruction is usually incomplete. Dehydration and fever are present if the child has not been treated with parenteral fluids.

X-rays of the abdomen show a dilated stomach and duodenum and only a few air bubbles in the remainder of the abdomen. Later the entire small gut may show dilatation. It is not necessary to use a contrast media to confirm the diagnosis, but if a barium meal is given a small amount of a thin mixture should be used so if regurgitation occurs the chances of aspiration pneumonia are lessened. The barium will usually advance to the obstructed portion of the duodenum and only a small amount will pass beyond this point. A barium enema will show the cecum in the epigastrium if malrotation is present.

Laboratory studies will show hemoconcentration, a high white blood count and a concentrated urine. An extremely high white blood count should lead one to suspect infarction of the midgut.

The conditions most often confused with malrotation are atresia and stenosis of the bowel and sometimes pyloric stenosis. As all of these are surgical emergencies no time should be lost for differential diagnostic purposes, and as soon as the diagnosis of obstruction is made laparotomy should be completed.

TREATMENT

The patient should be well hydrated and the electrolytic balance returned with glucose and saline. In emaciated patients small transfusions of whole blood are of great aid. A small gastric tube should be passed through the nose into the stomach before surgery.

The abdomen should be opened through a long paramedian incision extending from the xyphoid to below the umbilicus. If duodenal obstruction exists the ascending and transverse colon will present as the abdomen is opened and inspection will reveal the duodenal obstruction to be caused either by direct pressure of the cecum or by congenital bands running from the cecum, over the anterior duodenal surface, to the right posterior abdominal wall, and thus causing the obstruction. This condition is cured by an operation devised by Ladd. The right posterior parietal peritoneum is divided just to the right of the cecum until the entire duodenum is cleaned anteriorly and the cecum is mobilized so it may be placed in the left upper quadrant. This is sufficient to correct the condition and the abdomen is closed.

If a midgut volvulus is present, coils of reddish blue small gut will be present when the abdominal cavity is entered and the right half of the colon will not be visible. The entire small gut must be delivered from the abdominal cavity and the volvulus reduced by rotating the entire small gut, usually in a counterclockwise direction, enough times to reduce the twist. After this the cecum will lie in the right upper quadrant, and it is very important to sever its lateral peritoneal attachment and place the cecum and ascending colon in the left side of the abdomen. The entire bowel should be inspected for other anomalies.

The anesthetic of choice in these cases has been found to be open drip ether, with oxygen being administered by nasal catheter during the entire procedure.

The following case histories from the Independent Surgical Service at the Charity Hospital of Louisiana in New Orleans exemplify the above.

CASE REPORTS

Case No. 1. A seven month premature colored male, spontaneous delivery. Postnatal examination revealed no abnormalities. Child was placed in the nursery for premature infants and routine care started. It was noted that the patient vomited after practically every feeding. The vomitus was often bile stained. All oral feedings were stopped for three days, fluid balance being maintained by clyses, intravenous fluid, and blood transfusion. A smal catheter was passed into the stomach and a small amount of thin barium was injected into the stomach. X-ray and fluoroscopy showed the stomach and first portion of the duodenum to be markedly dilated and only a trickle

of barium was seen to enter the small gut. A three hour plate showed approximately 75 per cent gastric retention with a small amount of barium in the terminal ileum.

At operation, the cecum and ascending colon were in the epigastrium just inferior to the greater curvature of the stomach. Division of many thin vascular bands passing from the right upper posterior peritoneal mass across the descending duodenum to the cecum relieved the obstruction, and gas was seen to pass into the small gut. Inspection revealed no other gross anomalies. The abdomen was closed in layers with interrupted black silk #000.

For the first four postoperative days the child continued to vomit, but after that time retained all feedings and began to gain weight. The remainder of the postoperative course was uneventful.

This was a case of duodenal obstruction by congenital bands due to malrotation of the midgut which responded to surgical therapy.

Case No. 2. A nine day old colored male, with a chief complaint of jaundice and vomiting. He had vomited since his first feeding and for three days prior to admission had vomited not only after feeding but many times between feedings.

Physical examination revealed an emaciated, dehydrated infant with a distended abdomen and normal peristalsis.

Laboratory: Hemoglobin 13.3 gm. or 90 per cent; RBC 5,000,000; WBC 20,000; polys. 68; lymphocytes 32; no normoblasts; no erythroblasts; Prothrombin time, 100 per cent of normal. Kahn and Kline negative. Urine: albumin negative; sugar negative; acetone negative. Trace of urobilinogen. Ceph. flocc. 2-3-; van den Bergh-31.4; icteric index, 133. X-ray of abdomen showed marked distention of bowel with fluid levels in upper left quadrant.

Hospital Course: Given intravenous dextrose and saline on admission; nothing per os ordered. Could tolerate only small amounts of water by mouth after three days. Again started vomiting and abdomen became distended. Thick feedings were started and child vomited only once in twenty-four hour period. No stools since admission. Abdomen markedly distended. Under open drip ether the abdomen was opened through a right paramedian incision. As the peritoneum was opened, dilated loops of bluish red small gut presented. A volvulus of the entire small intestine was found consisting of two complete twists (720°). This was reduced and a mobile cecum was found. On inspection of the gut two gangrenous plaques were found in the midjejunum. This area was resected and a side-to-side anastomosis was done with two rows of interrupted quilting cotton. The abdomen was closed in layers with interrupted quilting cotton.

The patient was given intravenous fluid, blood, and sufadiazine by clyses. The temperature continued to rise, pneumonia developed, and patient expired on the third postoperative day. The pathology report on the resected gut was "moist gangrene of gut; stricture of gut due to chronic granulation tissue".

This case represents malrotation with a mobile, unattached cecum, and resulting volvulus of the small gut which caused infarction and gangrene. The pathology report of "stricture due to granulation" was due to the fact that the infarcted area had probably been present since admission and had begun to granulate. Death was due to peritonitis and pneumonia. The jaundice was explained by the postmortem finding of fibrosis of the pancreas.

Case No. 3. A ten day old colored female infant, who had been vomiting since birth presented a volvulus of the entire smal bowel at operation. Following reduction of volvulus the normal color returned to the bowel. The cecum and ascending colon were allowed to remain in the left side.

Patient developed a severe diarrhea on the seventh postoperative day but recovered. In contrast to Case No. 2, early surgery was successful.

Case No. 4. A four day old white male, admitted December 19, 1947, with a chief complaint of vomiting all feedings since the first twenty-four hours *X-ray of chest and abdomen*: "Suggestive atelectasis involving right upper and right middle lobes, with emphysema of left lung. Stomach gas bubble well demonstrated. There is only one other gas bubble just to the right of the spine. Findings are compatible with duodenal obstruction." *Hospital Course*: Patient put on penicillin, clyses, intravenous fluid and blood transfusion. *Repeat X-ray (one day later)*: "Clearing of rt. sided pneumonia. Abdominal findings unchanged."

Under drip ether and oxygen, the entire small bowel was delivered onto the abdominal wall and was found to have a 90 per cent twist on its meso. The cecum and appendix lay on the duodenum in the right upper quadrant attached by bands extending to the right lateral peritoneal gutter and obstructing the duodenum at this point. The bands were severed and the cecum moved to the left upper quadrant.

Patient vomited intermittently for several days, then began to retain all feedings well.

This case illustrates obstruction of the duodenum by congenital bands, plus an early volvulus of the small gut.

CONCLUSIONS

1. The embryology, etiology, diagnostic points, and treatment of congenital malrotation of the midgut have been briefly reviewed.

2. This is one of the congenital conditions which carries a good prognosis.

3. Early diagnosis and treatment are essential. The diagnosis can usually be made

from the history, physical examination, and regular EPA x-ray of the abdomen.

4. The case histories of four patients seen at Charity Hospital. of Louisiana in New Orleans, in the past five years are presented.

REFERENCES

1. Medaris, D. N.: Intestinal obstruction in the newly born due to an error in rotation of the midgut loop during fetal development, J. Kansas M. Soc. 46:328, 1945.
2. Rosenblatt, Millard S.: Congenital malrotation of loop of bowel with obstruction, Northwest Med. 45:336, 1946.
3. Bishop, Leutan H. Jr.: Congenital anomalies of the lower gastrointestinal tract causing obstruction, J. M. A. Georgia 36:223, 1947.
4. Steward, John A.: Faulty intestinal rotation, Am. J. Surg. 65:425, 1946.
5. Meade, H. S.: Reverse Rotation Producing Encapsulation of Small Intestine, Irish J. M. Sc. p. 581, 1944.
6. Ladd & Gross: Abdominal Surgery of Infancy and Childhood, W. B. Saunders Co., Chapter V.

—o—

THE CHANGING PICTURE IN APPENDICEAL DISEASE

A FIFTEEN YEAR SURVEY AT TOURO INFIRMARY*

GERALD N. WEISS, M. D.†

NEW ORLEANS

In March, 1934, Dr. Urban Maes delivered a paper on acute appendicitis before the Orleans Parish Medical Society in which he stated:

". . . the death rate . . .is almost as high today as it was nearly fifty years ago when Fitz, the internist, first described appendicitis as a surgical entity and Morton, the surgeon, first deliberately operated for it."

Despite the early excellent monographs of Kelly,[2] Deaver,[3] and Battle,[4] and the introductory classical articles by Fitz,[5] McBurney,[6] Murphy,[7] and other renowned surgeons, the mortality of appendicitis was at a record high of 15.2 per 100,000 population in 1930.[8a]

Within recent years, however, the mortality of this disease has fallen to 2.9 per 100,000 population in this country.[8d] Current articles,[9-11] indicate that the antibiotics, gastrointestinal tubal decompression, simplified and adequate methods for parenteral fluid and blood administration, early ambulation and the like, have contributed greatly to this decline in mortality. Equally as important as, if not more important than, all of these factors concerned with improved hospital care of the patient is the fact that the physician and the lay public are becoming aware of the grave situation created by delay in treatment of appendiceal disease. The continuing programs instituted in such cities as Philadelphia[12] and Cleveland,[13] and transient programs in New Orleans[14] and other cities have resulted in this education of the laity.

With the statistical picture in appendicitis greatly improved, a thorough analysis of the condition during the past fifteen years at Touro Infirmary leads one to feel that constant vigilance must be maintained and the latest facts reviewed to understand the changing picture in appendiceal disease. The fundamentals remain unchanged, but a new surgical generation is growing up that sees appendicitis as an almost benign disease; that sees only the great decrease in its mortality and overlooks its seriousness, a seriousness that was forced upon the older surgeon by the high fatality rate.

Appendiceal disease is no longer the medical problem of years ago, but it still merits periodic analyses and study. The trend in medical literature is clearly evident, as seen in the *New Orleans Medical and Surgical Journal*. Between the years 1934 and 1944, in this journal there appeared 26 articles ([1, 14-38]) concerned with the problems of appendicitis. In the following years there has been but 1 paper concerned with appendiceal disease and this dealt with a rare entity, carcinoid of the appendix.[39] The time is certainly ripe for another discussion of the subject, not only within medical circles but also among the lay public. Statistics continue to reveal that a degree of unawareness still exists among both medical and lay groups. Boyce[10] has recognized this in his current book on acute appendicitis, in which he says:

"Most of the deaths from acute appendicitis originate outside of the hospital and the surgeon, though he bears the greater responsibility, has the least to do with them, since the mortality is determined above everything else by the proportion

*Presented at meeting of the Orleans Parish Medical Society, February 13, 1950.

†Resident in Surgery, Touro Infirmary, New Orleans, Louisiana.

of patients admitted with appendices already ruptured."

In the case of the general practitioner, it is time to re-emphasize the statement of Osler,[41] that one

". . . does well to remember—whether his leanings be toward the conservative or radical methods of treatment—that the surgeon is often called too late, never too early."

CLASSIFICATION

The varied classifications of appendiceal disease have been responsible for a wide range of statistics concerned with mortality due to appendicitis. There has been no absolute classification, but the one recommended here has been prepared after reviewing the proved cases. These cases were confirmed by the pathologist's analysis of specimens removed either at the operating or on the autopsy table. Appendiceal disease at Touro Infirmary is coded in accordance with the system recommended by the American Medical Association in *Standard Nomenclature of Disease*.[42] The following classification seems to be most in accord with clinical application:

I. Appendicitis
  A. Acute
    1. Without complication
    2. With complication
      a) Gangrene
      b) Abscess
      c) Perforation
  B. Chronic
    1. Recurrent, active or inactive types
    2. Inactive or asymptomatic (incidental to other surgery)

II. Miscellaneous types of appendiceal disease
  A. Appendicopathia oxyurica
  B. Carcinoid
  C. Endometriosis
  D. Tuberculosis
  E. Adenocarcinoma
  F. Other Types

INCIDENCE

Appendiceal disease of all types is common. It constituted an average of 7.5 per cent of the 190,487 admissions to Touro Infirmary in the years 1934 through 1948. Of over 49,000 patients admitted to the general surgical service during this period, 17 per cent had proved appendiceal disease. Figure 1 illustrates the incidence of the

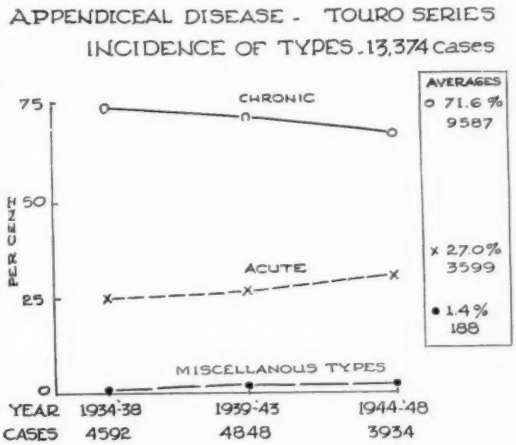

Fig. 1. Incidence of Types of Appendiceal Disease in 13,374 Cases (Touro Series)

types of appendiceal disease. Once almost three-fourths of the cases of appendiceal disease were chronic in nature. Now only two-thirds of all the cases fall into this group. Correspondingly, the incidence of acute appendicitis has risen proportionately from one-fourth to almost one-third. Miscellaneous types averaged less than 2 per cent of the total series.

The over-all mortality rate in 13,374 cases of proved appendiceal disease in the fifteen year period at Touro Infirmary was 0.7 per cent or 92 deaths. Figure 2 illus-

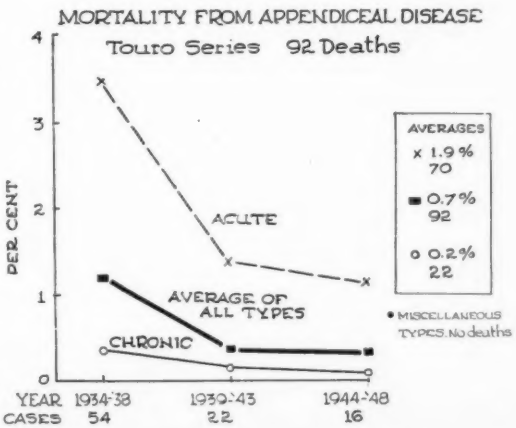

Fig. 2. Mortality from Appendicitis (Touro Series)

trates a reduction of two-thirds in the mortality of the disease in the five year period 1944 through 1948 as compared with the period 1934 through 1938. This reduction corresponds to that reported during the same period at other medical centers throughout the country and as reported by the National Office of Vital Statistics[8] (Fig. 3).

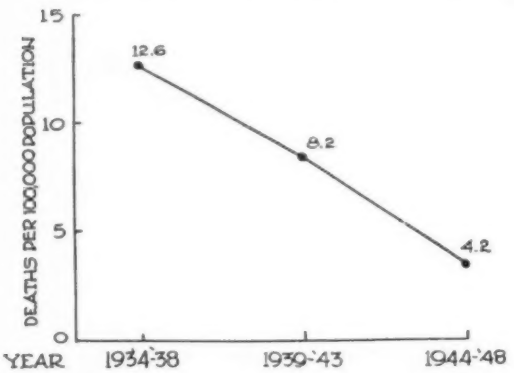

Fig. 3. Mortality from Appendicitis (National Office of Vital Statistics)

The necessity of classification into standard subgroups in the reporting of incidence and mortality statistics for appendicitis, which is not generally observed, is quite emphatically revealed in a study of this sort. For example, the mortality for appendiceal disease of all types was the result in about 40 per cent of the cases of acute appendicitis with perforation, a condition present in less than 2 per cent of the total series.

If appendicitis statistics are to be accurate, they must be classified, particularly with reference to the proportion of cases of perforation included in a series of acute appendicitis. Ochsner, Gage, and Garside, in 1930,[43] presented a table of comparisons showing that Clairmont and Meyer in a series of 1,594 cases had a mortality of 3 per cent whereas in the 193 cases in the authors' Charity Hospital of Louisiana series the mortality was over 20 per cent. The principal reason for this great discrepancy is the fact that in the first series of acute appendicitis only 0.24 percent of the patients had diffuse peritonitis, whereas in the Ochsner *et al* series the percentage was 11.9. When evaluating statistical analysis of acute appendicitis, it is obvious that the proportion, as well as the respective mortality of each type of acute appendicitis must be specified.

ACUTE APPENDICITIS

The 3,599 cases of acute appendicitis observed in the fifteen year period at Touro Infirmary carried a mortality of 1.9 per cent. Of course, in recent years the mortality figure has been much lower than the average; for example, during 1948, in 281 cases there was a mortality of only 0.7 per cent. In comparing the three five year periods (Fig. 4), it is clear that acute appendicitis without complication has increased from 70 to almost 90 per cent of the cases. Even more important is the fact that the incidence of gangrene, abscess, and perforation has fallen in the same proportion. This would indicate that earlier more accurate diagnosis and treatment is being instituted in appendicitis.

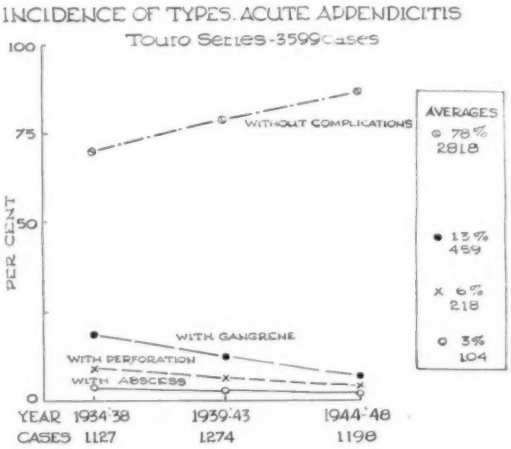

Fig. 4. Incidence of Types of Acute Appendicitis in 3,599 Cases (Touro Series)

Boyce's series of 6,441 cases from Charity Hospital of Louisiana at New Orleans can be compared with the 3,599 cases observed at Touro Infirmary in which more

private than indigent cases are seen (Table 1). It can readily be recognized that the

TABLE 1
ACUTE APPENDICITIS

Types	Charity Series—6441 Cases 1930-1945 Per Cent of Cases		Touro Series—3599 Cases 1934-1949 Per Cent of Mortality	
	Charity	Touro	Charity	Touro
Without Complication	51	78	0.6	0.4
With Gangrene	23	13	5.0	3.3
With Abscess	7	3	13.0	7.0
With Perforation	19	6	14.0	17.0
			5.0	1.9

5 per cent mortality in the Charity Hospital series is attributable primarily to the greater percentage of patients seen with the complications of acute appendicitis, notably perforation. This fact serves to emphasize the need of education of patients in the lower economic groups who are being seen late in the course of appendicitis. A campaign to educate these groups might bring the mortality of the charity and of the private institutions to a more equal standing.

Although the incidence of complications and mortality in the Touro series has been reduced greatly, an analysis of the deaths shows many significant facts (Table 2). Over one-half of the deaths from acute appendicitis are now occurring in the age group over 40. Procrastination and purgation are still the principal factors contributing to death. Peritonitis remains the primary cause of death in over three-fourths of the cases. Over one-third of the patients who died gave a history of at least one previous attack.

In examining the problem of delay more closely, it is noted that in the 70 deaths from acute appendicitis, almost one-third of the histories revealed delay of over twenty-four hours by the patient and a somewhat greater percentage delayed on advice of a physician. In one-sixth of the cases the reason for delay was not specified, although 19 per cent of the total patients were from out of town and difficulty in transportation perhaps was a factor. The small remaining group of patients applied for treatment within the twenty-four hour period. Fifteen years ago, Maes[22] reported an almost equal proportion of deaths (i. e. 79 per cent) with a delay of over twenty-four hours in treatment. Procrastination, therefore, as in years past, is still the primary contributing factor in death from appendicitis. Delay by the patient definitely shows lack of knowledge of the disease, and perhaps in some cases can be attributed to difficulty in transportation. But why in over one-third of the 70 deaths was there delay by the physician? Either the physician did not diagnose the disease correctly, or he did not recognize it as an acute surgical emergency. Certainly, all physicians should be aware of the facts as stated by Murphy:[7]

"From the symptoms and clinical course of the disease in the first forty-eight hours it is impossible to predict with any degree of certainty, what the course of the case will be . . ."

It is mandatory, therefore, when the diagnosis of acute appendicitis is made, or cannot be safely ruled out, that early surgical therapy is indicated, for the danger in delay is far more serious than the appendectomy. And now, almost one-half a century since Murphy's classic article on appendicitis, one too must conclude as did he:

"It seems to me that every death from appendicitis is chargeable directly to the people, either for not calling in the physician sufficiently early after the onset of symptoms, or to the physician and surgeon for not acting promptly when called. I am sorry to admit that the latter represents the greater percentage . . ."

One is tempted to question at this point, would not deaths from acute appendicitis be less common if the incidence of the treatment of chronic recurrent appendicitis had not fallen? In any event, 37 per cent of the patients who died gave a history of at least one previous attack (Table 2). It is probable that interval appendectomy would have saved these patients.

Purgation, too, remains a major factor responsible for deaths from appendicitis. In Maes' series[22] 49 per cent of the patients who died were reported to have taken laxatives. In the present series, 27 per cent, or over one-fourth took a purgative with the attack. Some improvement can be recorded in reference to this aspect of the

TABLE 2
DISTRIBUTION AND MORTALITY OF 3599 CASES OF ACUTE APPENDICITIS AT TOURO INFIRMARY 1934-1948

	Without Complication	With Complication: Gangrene	Abscess	Perforation	Total Cases	Per cent of Total	
Total Cases	2818	459	104	218	3599	.....	
Total Deaths	11	15	7	37	70	100	
Sex: Male	5	12	4	22	43	61	
Female	6	3	3	15	27	39	
Age under 13	1	0	1	7	9	13	
13-39	3	3	2	14	22	31	
Over 39	7	12	4	16	39	56	
Out of town	2	2	2	7	13	19	
Onset less than 24 hrs.	6	4	0	4	14	20	
over 24 hrs.	5	11	7	33	56	80	
Previous Attack	6	4	5	11	26	37	
Laxative with Attack	1	4	0	14	19	27	
Enema with Attack	2	3	0	3	8	11	
Delay over 24 hrs.							
Patient	5	3	2	10	20	28	
Physician	1	2	5	17	25	36	81
Unspecified	0	6	0	6	12	17	
Failure of diagnosis on hospital admission	2	3	5	13	23	33	
Surgery Yes	10	15	6	26	57	81	
No	1	0	1	11	13	19	
Peritonitis at surgery							
Localized	0	8	4	5	17	30	
Generalized	2†	7	3	21	33	58	88*
Peritoneal Drainage	0	3	6	21	30	53*	
Incisions							
Deaver	3	9	3	10	25	44	
McBurney	4	4	0	9	17	30	
Battle	1	1	0	5	7	12	
Midline	1	1	1	0	3	5	100*
Transverse	1	0	0	2	3	5	
Kidney	0	0	1	0	1	2	
Left Paramedian	0	0	1	0	1	2	
Anesthesia							
General	6	11	3	20	40	70	
Spinal	4	4	2	5	15	26	100*
Local	0	0	1	1	2	4	
Deaths from Peritonitis and Complications	4	7	7	37	55	79	
Other Causes	7	8	0	0	15	21	

†Technical error at time of surgery resulting in generalized peritonitis
*Percentage of the 57 operative cases

problem. It is clearly evident, however, that the lay public is still not sufficiently educated to the danger of the use of laxatives or high enemas with abdominal pain.

The principal cause of death from acute appendicitis continues to be peritonitis. Fifty-five of the 70 deaths, or 79 per cent of the cases, died with this terminal complication. The last local report on the mortality of acute appendicitis,[11] fifteen years ago, revealed a comparable mortality.

It is interesting to examine the reason for failure to operate in one-fifth of the deaths due to acute appendicitis (Table 2). In the 13 cases not treated surgically, the omission of operation was due in 7 cases to error in diagnosis by the physician, in 3 cases to the institution of conservative therapy, and in the remaining 3 cases to refusal of surgery by the patient. In 8 of

these cases, well over half, the patients were in the age group over 40 and atypical symptomatology made diagnosis difficult.

Figures gathered in this series of deaths as to the type of anesthesia, incision, and drainage do not in themselves prove to be illuminating. The consensus of current literature[13, 40, 44] is toward a preference for spinal anesthesia, anatomicophysiologic incisions (McBurney or transverse), handling of the appendiceal stump by purse string inversion with or without ligation, and less frequent drainage of the peritoneal cavity since the advent of antibiotics.

CHRONIC RECURRENT APPENDICITIS

Coded under chronic recurrent appendicitis were 9,587 cases with a mortality of 0.2 percent. During the past ten years there has been a steady decline in the number of these cases, so that during the period 1944 through 1948, only slightly more than two-thirds of the cases of appendiceal disease were thus classified.

It became obvious in reviewing the deaths classified and coded according to the hospital requirements as chronic recurrent appendicitis, that two distinct groups existed. Appendices might be removed and sent to the pathologist with a clinical diagnosis of chronic recurrent appendicitis, while others might be removed incidentally in the course of another abdominal procedure. Both appendices would show similar pathology and so a diagnosis of "chronic appendicitis" would be reported by the pathologist. The resident staff, in completing the patient's record, sign out all such cases as chronic recurrent appendicitis, as no provision is made for the category of chronic appendicitis, asymptomatic or inactive. No attempt was made to analyze these cases closely; but a study of the deaths was made and in one-half of the 22 fatalities indexed as chronic recurrent appendicitis a concommitant surgical procedure was found to have been performed. The 11 deaths following appendectomy alone were attributable in 5 cases to peritonitis, in 3 cases to pulmonary complications, and in 1 case each to septicemia, coronary disease, and mesenteric thrombosis. It would seem that some of these deaths might have been prevented. In 2 instances had an incision of more than 2½ inches been used, difficulties and subsequent death might have been avoided. These deaths serve to emphasize that although the fatality rate for chronic recurrent appendicitis has recently been reduced to less than 1 in 1000 cases (Fig. 2), no operation can ever be regarded lightly or performed in a compromising fashion.

The remaining deaths in which the appendix was removed and found to be chronically diseased were associated with the following conditions and additional surgery: 6 cases with galbladder disease, 3 cases with hysterectomy, and 1 case each of intestinal obstruction and tubal sterilization. Incidental appendectomy in no case was the primary cause of death in these instances.

It is exceedingly rare that incidental appendectomy is responsible for an operative or postoperative fatality. On the other hand, there are many cases in which, if the surgeon while operating within the abdominal cavity would practice prophylactic surgery, the patient might be spared another operation or even saved from death.

MISCELLANEOUS TYPES OF APPENDICEAL DISEASE

There were 188 cases classified in the category of miscellaneous types of appendiceal disease, without a death. Over half of the cases were oxyuriasis of the appendix or so called "appendicopathia oxyurica".[45] Next in order of frequency were the carcinoids (15 per cent), endometriosis (10 per cent), tuberculosis (4 per cent), and adenocarcinoma (4 per cent). The remaining 15 per cent of the total were divided among the following diseases: helminthiasis, diverticulum, mucocele, foreign body, ascariasis, intussusception, transposition, congenital absence, carcinoma, neuroma, and neurofibroma.

It is interesting that in this period there was not a single death from miscellaneous types of appendiceal disease, since all of them occurred with either acute or chronic appendicitis.

SUMMARY

The incidence of hospital admissions to Touro Infirmary of New Orleans for appen-

diceal disease was 7.5 per cent of 190,487 admissions in the fifteen year period 1934 through 1948.

The mortality for appendiceal disease has fallen by two thirds with an average fatality rate in 3,374 cases of 0.7 per cent, or 92 cases.

In the series of 3,599 cases of acute appendicitis there was a death rate of 1.9 per cent. Approximately:

1 in 250 patients died of acute appendicitis without complication.

1 in 30 patients died of acute appendicitis with gangrene,

1 in 15 patients died of acute appendicitis with abscess, and

1 in 6 patients died of acute appendicitis with perforation.

In the series of 9,587 cases with chronic appendicitis, both recurrent and those removed incidental to other surgical procedures, the mortality was 0.23 per cent, or approximately 1 in 430 patients died.

Although earlier more accurate diagnosis and treatment are being instituted in appendicitis, mortality is due in great part to the same factors which were responsible in years past, procrastination and purgation.

Interval appendectomy for patients with a history of one or more attacks of appendicitis, and appendectomy where feasible during the course of other intra-abdominal operations will aid in reducing mortality from appendiceal disease.

Periodic analyses of the problem of appendiceal disease are indicated and should be presented to medical and lay groups at regular intervals.

REFERENCES

1. Maes, Urban, Boyce, F. F., McFetridge, E. M.: Acute appendicitis between the extremes of life with analysis of 910 cases, New Orleans M. & S. J. 87:24, 1934.

2. Kelly, Howard A.: Appendicitis and Other Diseases of the Vermiform Appendix, Philadelphia, J. B. Lippincott Co., 1909.

3. Deaver, John B.: Appendicitis, Its History, Anatomy, Clinical Aetiology, Pathology, Symptomology, Diagnosis, Prognosis, Treatment, Technique of Operation, Complications and Sequels, Philadelphia, P. Blakeston's Sons and Co., 1905.

4. Battle, Wm. Henry and Corner, Edred M.: The Surgery of the Diseases of the Appendix Vermiformis and their Complications. Chicago, W. T. Keener and Co., 1905.

5. Fitz, Reginald H.: Perforating inflammation of the vermiform appendix with special reference to its early diagnosis and treatment, Tr. A. Am. Phys. 1:107, 1886.

6. McBurney, Charles: The incision made in the abdominal wall in cases of appendicitis, with a description of a new method of operating, Ann. Surg. 20:38, 1894.

7. Murphy, John B.: Two Thousand Operations for Appendicitis with Deductions from His Personal Experience, Am. J. M. Sc., 128:187, (Aug.) 1904.

8. National Office of Vital Statistics, Special Reports, Washington, D. C.
   a) Mortality Summary for U. S. Registration States, Appendicitis, Vol. 16, No. 33, page 133, (Sept. 12) 1942
   b) Mortality Summary for the United States, Appendicitis, Vol. 27, No. 45, (Mar. 15) 1948.
   c) Deaths and Death Rates for Each Cause: United States 1945-47, Vol. 31, No. 3, (May 9) 1949.
   d) Estimated Numbers of Deaths and Death Rates for Specified Causes, United States, 1948, Vol. 6, No. 13, (Sept. 16) 1949.

9. Lehman, E. P., Hawk, J. C., and Becker, W. F.: Appendicitis, with consideration of the new drugs in appendiceal peritonitis, S. Clin. N. America, 1385, (Oct.) 1949.

10. Farris, J.: The effect of streptomycin in 'Closed Loop' Appendicitis, Surgery 22:305, 1947.

11. Hoerr, Stanley O.: Factors in the reduction of mortality in acute appendicitis, Surgery 22:402, 1947.

12. Bower, J. O.: Mortality of Acute Appendicitis: report of progress made in campaign for its reduction in Philadelphia, J. A. M. A. 99:1765, (Nov. 19) 1932.

13. Green, Howard W. and Watkins, Ralph M.: Appendicitis in Cleveland, final report, Surg., Gynec. & Obst. 83:613, 1946.

14. The mortality of acute appendicitis in the hospitals of New Orleans: a report, New Orleans M. & S. J. 89:170, 1936.

15. Friedrichs, Andrew V.: The etiology and pathology of appendicitis, New Orleans M. & S. J. 87:20, 1934.

16. Mattingly, C. W., Cecostomy in the treatment of the ruptured appendix and peritonitis, New Orleans M. & S. J. 87:31, 1934.

17. Ochsner, Alton: The conservative treatment of appendiceal peritonitis, New Orleans M. & S. J. 87:32, 1934.

18. McBride, D. C.: Major facts concerning appendicitis, New Orleans M. & S. J. 87:215, 1934.

19. Culpepper, J. P., Jr.: Appendicitis during pregnancy, New Orleans M. & S. J. 87:370, 1934.

20. Wolford, Thomas: Retrocecal appendices, New Orleans M. & S. J. 87:622, 1935.

21. Shute, F. C.: The perforated appendix, New Orleans M. & S. J. 88:171, 1935.

22. Maes, Urban, Boyce, F. F., and McFetridge, E. M.: The mortality of acute appendicitis, an analysis of 156 fatalities, New Orleans M. & S. J. 89:118, 1936.

23. Signorelli, John: Acute appendicitis: A symposium, Acute appendicitis in children, New Orleans M. & S. J. 89:156, 1936.

24. Bethea, Oscar W.: Medical aspects of appendicitis, New Orleans M. & S. J. 89:162, 1936.

25. Daver, C. C.: Appendicitis as a community health problem, New Orleans M. & S. J. 89:164, 1936.

26. Storck, Ambrose: The part played by the surgeon in lowering the mortality of acute appendicitis, New Orleans M. & S. J. 89:165, 1936.

27. Boyce, F. F. and McFetridge, E. M.: The presentation of acute appendicitis in standard text-books and systems, New Orleans M. & S. J. 89:167, 1936.

28. Fortier, L. A., and Gately, T. T.: X-rays in chronic appendicitis, New Orleans M. & S. J. 89:200, 1936.

29. Pratt, J. P.: Pelvic conditions simulating appendicitis, New Orleans M. & S. J. 90:183, 1937.

30. Coon, Henson S.: The use of an anatomicophysiologic incision in appendicitis, New Orleans M. & S. J. 92:135, 1940.

31. Cohn, Isidore: The aims of the appendicitis committee of the surgical section of the Southern Medical Association, New Orleans M. & S. J. 92:489, 1940.

32. Kahle, H. R.: The diagnosis of acute appendicitis, New Orleans M. & S. J. 92:490, 1940.

33. Karlin, Samuel: The management of the patient with possible appendicitis, New Orleans M. & S. J. 92:494, 1940.

34. Miangolarra, C. J.: Age factors in acute appendicitis, New Orleans M. & S. J. 92:496, 1940.

35. Ledoux, Lucien A.: Acute appendicitis in the female, New Orleans M. & S. J. 93:140, 1940.

36. Boyce, Frederick F.: The mortality of acute appendicitis, New Orleans M. & S. J. 93:300, 1941.

37. Nix, James F. and Nix, J. T., Jr.: Left sided appendicitis, New Orleans M. & S. J. 94:530, 1942.

38. Boyce, F. F.: The diagnostic confusion between acute appendicitis and pelvic inflammatory disease, New Orleans M. & S. J. 96:303, 1943.

39. D'Ingianni, Vincente: Carcinoid of the appendix with metastasis, New Orleans M. & S. J. 99:158, 1946.

40. Boyce, Frederick F.: Acute Appendicitis and Its Complications, New York, Oxford University Press, 1949.

41. Osler, Sir William quoted by Sloan, Harry G., "Appendicitis", Lewis' Practice of Surgery, edited by Walters, Waltman, Vol. 7, Hagerstown, Maryland, W. F. Prior Company, 1949.

42. Standard Nomenclature of Disease, edited by Jordan, Edwin P., (published for the American Medical Association), Philadelphia, The Blakiston Co., 1942.

43. Ochsner, Alton, Gage, I. M., and Garside, Earl: The intra-abdominal postoperative complications of appendicitis, Ann. Surg. 91:544, (April) 1930.

44. Ochsner, Alton, and Barrow, David W.: Appendicitis: Its Surgical Treatment, Surgical Treatment of the Abdomen, edited by Bancroft, Frederic W., and Wade, Preston A., Philadelphia, J. B. Lippincott Co., 1947.

45. Aschoff, L.: Med. Klin. 9:249, 1913, quoted by Anderson, W. A. D., Pathology, St. Louis, C. V. Mosby, 1948.

DISCUSSION

Dr. Urban Maes (New Orleans): In May 1936, the Orleans Parish Medical Society put on, as a part of its program, what they chose to call "Longer Life Week". This was part of an educational program which was contemplated as an annual feature. The subject at that time was acute appendicitis and brief talks were made at hospitals, schools, clubs, and other gatherings, and special emphasis was laid on the dangerous administration of purgatives in acute abdominal conditions. I was asked to be the chairman of the committee and used as a slogan—procrastination and purgation were the two greatest factors in the death rate from acute appendicitis. These points have been brought to our attention again in this statistical study by Dr. Weiss.

At that time we analyzed 1848 cases collected from several hospitals and found the overall death rate to be 5.8 percent or 107 deaths in 1848 patients. In this series the death rate when purgatives had been administered was over 10 per cent or nearly double. The time element also played an important part. When patients were cared for in the first twenty-four hours, the death rate was 2.1 per cent, whereas at the end of twenty-four hours the rate was 8.7 per cent, and when forty-eight hours had elapsed the rate was 11.7 per cent. Our figures regardless of the time element show that a person who has taken a purgative is twice as likely to succumb. The purgative had been ordered by the physician in only 10 patients.

Dr. Weiss has shown by his figures that the death rate has been reduced by two-thirds, and in analyzing his records the complications also have been materially reduced. There are three obvious reasons for this salvage:

1. Earlier treatment is noticeable.
2. The withholding of purgatives is noteworthy.
3. The use of antibiotics has reduced the incidence and helped the control of peritonitis.

It may be that the educational program did some good and the laity has shown more wisdom in the indiscriminate use of purgative for acute abdominal pain. This and earlier operation even in doubtful cases has undoubtedly saved many lives.

Acute appendicitis is not a dangerous disease when the disease process is confined to the lumen of the appendix. It is only when rupture, gangrene, or perforation have occurred that acute appendicitis becomes a dangerous disease. In other words, the complications that kill are extra-appendicular. The remedy is obvious.

It is interesting to note that Dr. Weiss reviewed 27 papers on this subject published in the last fifteen years, and 26 of these are over ten years old. Probably the educational program did some good.

—o—

CARDIAC PREDECOMPENSATION*

EDGAR HULL, M. D.

NEW ORLEANS

Under the term *cardiac predecompensation*, I shall include two types of cases: (1) Persons with organic heart disease, who have no symptoms or signs of decrease of myocardial reserve; and (2) persons with heart disease who have symptoms and signs due to cardiac dysfunction but who as yet have not developed frank heart failure.

The first category is of particular importance to the general practitioner, for this is the group in which heart disease is discovered as an incidental finding in patients who visit the physician for a general check-up or for the relief of unrelated complaints. Although, as a general rule, patients with asymptomatic heart disease require no treatment directed at their heart disease, it is important that such patients be examined at intervals so that incipient cardiac decompensation may be recognized in its earliest stages, and in certain cases something can be done about the heart disease as such. For example, in syphilitic heart disease, which on occasion may be

*Presented at the Second Annual Scientific Session, Louisiana Academy of General Practice, May 7, 1949, in New Orleans.

picked up in the early stages, when one hears an accentuated aortic second sound or an aortic diastolic murmur, careful and thorough antisyphilitic treatment may prevent further progression of the lesions so that heart failure may be entirely prevented.

Again, certain asymptomatic congenital anomalies, such as coarctation of the aorta and patent ductus arteriosus, if recognized in childhood, may actually be cured by surgical measures, and the cardiovascular system restored practically to a normal state.

Hyperthyroidism, myxedema, and diabetes, conditions which may eventually cause heart disease and heart failure, may be prevented by proper treatment, and anemia and vitamin B deficiency are other examples of conditions which affect cardiac function and which are readily amenable to treatment.

Hypertensive heart disease in the stage of predecompensation is less amenable to treatment because certain effective measures for the control of hypertension have not as yet been discovered, but in some cases it may be possible to reduce the blood pressure and thus decrease the burden of the heart.

Constrictive pericarditis, which produces a very definite and easily recognizable syndrome, is amenable to surgical treatment, but such treatment is usually not indicated unless severe symptoms have resulted from compression of the heart.

It behooves the general practitioner, therefore, to be familiar with the symptoms and signs of these conditions in their presymptomatic stages, so that if possible the patient may either be removed from the category of cardiac predecompensation or remain in this category indefinitely and never develop frank cardiac failure. We shall, however, spend most of our time today discussing the recognition and treatment of the earliest stages of cardiac insufficiency, stages in which therapy is usually quite effective, so that if treatment is begun at this time, frank cardiac failure may be prevented for many years or even indefinitely. Again, patients with such early cardiac decompensation are usually seen first by the general practitioner, as indeed are patients with most diseases and conditions in their earliest stages, so that the responsibility for prolonging the useful lives of these individuals falls principally on him.

The great majority of patients in this category of cardiac predecompensation will be middle-aged or elderly individuals with hypertensive or arteriosclerotic heart disease; a small number will have rheumatic heart disease, and an even smaller number heart disease of other etiology.

SUBJECTIVE SIGNS

The subjective symptoms of early or mild decompensation are due principally to two causes:

1. Decrease of cardiac output, which results in subnormal blood flow through the body, and which in the early stages is manifested principally by easy fatigability, muscular weakness, and sometimes by cerebral symptoms such as dizziness, inability to concentrate, nervousness, and irritability. These symptoms are especially apt to apear in the afternoon or evening, after the ordinary activities of the day, for in most patients with early cardiac failure the cardiac output is not greatly reduced below normal at rest.

2. Passive congestion, due to backward failure and increase of blood volume, is manifested principally by dyspnea on exertion, upper abdominal discomfort—especially marked after meals, slight enlargement of the liver, slight or moderate distention of the neck veins, and in some cases puffiness of the feet and ankles toward afternoon.

There is considerable variation in the subjective complaints of patients with incipient cardiac failure. Some will complain only of easy fatigability and indigestion, for example; others will have as their chief complaint right upper quadrant pain related to congestion of the liver; still others will complain of dyspnea on exertion. Since all of these complaints may be due to factors other than diminution of myocardial

reserve, determination of their exact pathogenesis is a matter of great importance.

OBJECTIVE SIGNS

In such an evaluation, one must rely almost entirely upon objective data, of which the most important by far are physical signs and x-ray signs. Of these, the following are of the greatest importance:

1. Enlargement of the heart, as manifested by displacement of the apex beat or by the x-ray. For practical purposes, it may be said that the absence of cardiac enlargement practically rules out the heart as the cause of such symptoms as those which have been mentioned.

2. Signs of pulmonary congestion. The most important physical sign of pulmonary congestion is the occurrence of rales at the bases of the lungs, but it should be emphasized that rales constitute a sign of pulmonary edema, and that a significant degree of congestion may be present without the occurrence of rales. The x-ray affords much earlier evidence of pulmonary congestion, manifested by exaggeration of the lung markings, especially in the region of the hilum. The combination of cardiac enlargement and exaggerated vascular markings is practically pathognomonic of pulmonary congestion due to cardiac decompensation.

3. Other physical signs which have been mentioned, such as venous distention, palpability of the liver, and pretibial edema.

The electrocardiogram plays no part in the recognition of cardiac decompensation, since this reveals only disturbances of impulse production or impulse conduction. It should be noted in this connection, however, that certain cardiac arrhythmias, such as auricular fibrillation, paroxysmal tachycardia, and even frequent premature beats, which can usually be recognized without recourse to the electrocardiogram, may impair cardiac efficiency and may thus predispose to or aggravate incipient cardiac decompensation.

It cannot be too strongly emphasized that cardiac decompensation is not the sole cause of dyspnea in middle-aged or elderly patients. Such dyspnea is frequently due to pulmonary or bronchial disease, and for this reason an x-ray of the chest is indicated in all patients who are short of breath. In some cases it will be found that the dyspnea is due to carcinoma of the lung or other serious pulmonary disease, while in others it will be found that pulmonary emphysema is the cause of the shortness of breath. In most cases pulmonary emphysema can be recognized without recourse to the x-ray, the principal physical sign being characteristic change in the shape of the chest. The x-ray findings are, however, quite characteristic, and in most such cases it will be found that the heart is not significantly enlarged. The history is very important, most patients with pulmonary emphysema complaining first of dyspnea on exertion, which is only very slowly progressive over the years, and which is later manifested by cough, mucopurulent or purulent sputum, wheezing, and the occurrence of attacks of wheezing dyspnea very similar to bronchial asthma.

TREATMENT

The treatment of cardiac predecompensation is, we may say, practically identical qualitatively with the treatment of frank heart failure, varying only in its quantitative aspects. These patients need rest and digitalization, and many of them need salt restriction and diuretics. However, rather than bed rest, they need moderate restriction of activity, with a short period of bed rest during the middle of the day, slow rather than rapid digitalization, moderate rather than drastic salt restriction, and small rather than large doses of mercurial diuretics. Cardiac arrhythmias in these cases may be controlled by slow rather than rapid quinidinization. A few details regarding the employment of these methods and medicines may be in order:

1. Rest. A good rule regarding the amount of activity allowed these patients is that activity should be less than the amount which will produce signs of backward or forward failure, principally dyspnea and fatigue. If the patient experiences such symptoms, he is exercising too much, but there is good evidence that exercise

short of the amount which will produce symptoms is beneficial rather than the reverse. In most cases it will be found that after the institution of other measures, the exercise tolerance of these patients will increase, and it is always safe to allow exercise which does not reach the tolerance point. Strenuous exercise, particularly sudden, strenuous exertion, is always to be avoided.

2. Digitalis. In my opinion, digitalization is indicated for practically all patients in this category, and in many cases it will be found that restriction of activity and digitalization are the only measures which are necessary. There is no point in rapid digitalization in such cases, since no emergency is present, and since the safety of digitalis administration undoubtedly varies inversely with the speed of digitalization. As a rule, USP digitalis is preferable to any of the purified preparations, a satisfactory method of its use being to give 1½ gr., three times a day for a week or ten days, followed by a maintenance dose of 1½ to 3 gr. daily for an indefinite period of time. We have found that most patients tolerate 3 gr. a day very well, without symptoms of digitalis intoxication, and that in many patients digitalis effect is lost on the usual smaller maintenance dose of 1½ gr. a day.

A few patients will not tolerate therapeutic doses of USP digitalis, complaining principally of anorexia, nausea, and upper abdominal discomfort, and for such patients one of the purified preparations is indicated. In such instances digoxin or digitoxin may be used, my own preference being digoxin because of its more rapid elimination and therefore the lesser likelihood of prolnged manifestations of digitalis toxicity. If digitoxin is used, it should be remembered that the ordinarily recommended digitalization dosage of 1.2 mg. is too small for most patients, while on the other hand the recommended maintenance dosage of 2 mg. is usually too large, so that a considerable number of patients will develop signs of intoxication after they have been taking this amount for more than a few weeks.

3. Salt restriction and diuretics. I think it is important to point out that measures which reduce the salt content of the body are valuable only in the control of passive congestion, and that they are therefore not indicated in patients whose symptoms are due only to forward failure. If there is no dyspnea, no hepatic enlargement, or no edema, and the patient complains only of fatigability or weakness or dizziness, salt restriction and diuretics, which result in decrease of blood volume, are apt to aggravate rather than to relieve the symptoms. There can be no doubt that the increase of blood volume in patients with cardiac decompensation serves a useful purpose, in that it enables weakened hearts to maintain a more nearly adequate output, and it is only when the increase of blood volume is excessive that it is necessary or admissible to attempt to reduce the amount of salt in the body. However, if symptoms or signs of passive congestion are present, salt restriction and diuretics are of definite value.

For the patient with minimal signs of congestion, only moderate restriction of salt intake combined with the use of ammonium chloride may be necessary. For other patients, the judicious use of mercurial diuretics is definitely indicated. I should say that if definite edema is present, or if mild signs of congestion persist after digitalization, salt restriction and the use of ammonium chloride, one of the mercurial diuretics should be employed. One is about as good as another, salyrgan-theophylline and mercuhydrin being somewhat less irritating when given intramuscularly than is mercupurin. These drugs should always be given intramuscularly, never intravenously; their diuretic effect is just as marked by intramuscular administration, and there have been more than a few instances of sudden death following immediately upon their intravenous use.

There is no need to give these drugs in large doses at very frequent intervals, and indeed there is danger attendant to rapid dehydration and salt depletion which may result from drastic diuresis. We have seen

several cases, and numerous others have been recorded in the literature in which severe symptoms or even death have resulted from the administration of large doses of mercurial diuretics at frequent intervals. Ordinarily, in cases of predecompensation, 1 cc. twice a week will suffice gradually to reduce the amount of water and salt in the body. It would be a good rule to weigh the patient when the course of mercurial diuretics is begun, and to continue their use until edema has disappeared and dyspnea is relieved, at the end of which time the patient should again be weighed. This may be labeled as normal weight, and the patient should thereafter weigh himself a couple of times a week, and an additional course of diuretic be given if his weight should increase more than 2-3 lbs. above this figure. Some patients will require no further injections for a period of months, whereas others may require injections once a week or once every two or three weeks.

It is to be noted that during the hot months, when patients may perspire a great deal and thus lose a considerable amount of salt, the danger of dehydration and salt depletion is increased.

I do not intend to deprecate the value of mercurial diuretics, for there is no doubt at all that their introduction represents the greatest advance in the treatment of heart failure since the introduction of digitalis by Withering one hundred and fifty years ago. Even after the development of frank congestive failure, restriction of activity, digitalization, and the judicious use of diuretics may enable patients to live comfortable and useful lives for several years; whereas before the introduction of mercurial diuretics, most patients who developed heart failure were dead within two years. Although in my opinion Dr. Harry Gold has advocated much too drastic use of mercurial diuretics, there is no doubt that his concept of maintenance doses of mercurial diuretics as well as maintenance doses of digitalis, has been extremely useful in the postponement and prevention of recurrences of congestive heart failure.

Time will not allow a discussion of the use of quinidine for the therapy of cardiac arrhythmias in the absence of frank heart failure, nor will there be time to discuss the symptomatic management of these patients. I have found, however, that the general practitioner usually needs no instruction about the judicious use of sedatives, and about the importance of caloric restriction in patients who are too fat, and especially about the informal psychotherapy which these patients need and which the family doctor is in much better position to give because he actually knows his patients as people, and is much better able to treat them as patients rather than as cases.

If the principles and the few details which I have given are followed, and if the importance of regular though not necessarily very frequent observation is recognized, there is no doubt at all that the general practitioner is in a better position to handle most patients with heart disease than is the internist or cardiologist.

DISCUSSION

Question: (Dr. J. M. Bodenheimer): What about intravenous use of arsenicals in heart disease?

Answer: Arsenicals are not contraindicated for patients with syphilitic heart disease provided their use is preceded by more gentle antisyphilitic treatment with iodides and bismuth. At present, for the treatment of syphilitic aortitis we recommend iodides, bismuth, mapharsen and penicillin in succession.

Question: (Dr. J. M. Bodenheimer): What about vitamin E?

Answer: I think it is pretty well established that it is of no value in short-term treatment of heart disease.

Question: (Dr. J. M. Bodenheimer): What about absorption of mercurial diuretics in marked edema if given intramuscularly?

Answer: That is a good question. Our experience indicates that there is usually adequate absorption if the diuretic is given deeply in the muscle.

Question: (Dr. F. H. Davis): Are there any particular precautions to follow or helpful hints in the use of some of the newer digitalis-like drugs?

Answer: So far as we are able to find out there is no evidence of any one of the purified glycosides having a superior effect to the leaf. The indications for the purified preparations would be principally in those cases of the occasional patient who won't tolerate the leaf by mouth in therapeutic doses in which case digoxin or digitoxin should

be used; and secondly, in emergency when rapid digitalization may mean life or death.

Question: (Dr. F. H. Davis): How about pulling of teeth, minor operations, etc. in these cases.

Answer: In my experience I have found patients with heart failure or frank decompensation can stand minor surgery well. There is no contraindication to minor operations in patients with myocardial predecompensation. If they respond to therapy major surgery can be performed with reasonable safety.

Question: (Dr. F. H. Davis): Should one use sulfa drugs in case of pulling teeth in these patients?

Answer: I would say whenever teeth, which are carious, are removed from sockets at the seat of pyorrhea, penicillin should be employed more or less routinely to prevent bacteremia whether there is heart disease or not. Following extraction of carious teeth or severe pyorrhea 20 or 30 per cent have transient bacteremia after the teeth are removed. I would not like to have transient bacteremia even if I did not have heart trouble.

Question: (Dr. Koretzky): What about the use of penicillin in luetic heart disease in patients past sixty years? Does heart size diminish with penicillin?

Answer: If the patient is past sixty and has not developed failure, I don't think he needs any treatment but there would be no contraindications in the aged individual. Most patients with syphilis past fifty or sixty years of age don't need any treatment.

———o———

TREATMENT OF THE COMMON COLD*

OSCAR W. BETHEA, M. D.
NEW ORLEANS

GENERAL CONSIDERATIONS

This discussion of the common cold, naturally, will not include such conditions as epidemic influenza or so-called virus pneumonia. It also does not seem practical or advisable to cover the many possible complications arising from secondary invasion of bacteria.

The medical and economic importance of colds cannot be overemphasized. "The common, acute, self limiting, chiefly air borne infections of the respiratory tract constitute the majority of conditions encountered in civilian practice. Thirty-two per cent of home visits and 15 per cent of office visits of the general practitioner are due to these conditions. There are about 250 million respiratory infections in the United States each year. Loss of time from industry and school resulting from these is more than from all other conditions combined."[1] It was one of the chief causes for admission into the medical wards of service hospitals during the last war.[2]

In the majority of instances the initiating agent belongs to the group of filtrable viruses. Organisms, such as the pneumococcus or streptococcus, take advantage of the preceding pathological changes and give rise, secondarily, to the more serious infections, such as sinusitis, otitis, bronchitis, and bacterial pneumonia.

While it is now generally acknowledged that the common cold is a virus infection,[3] electron microscopic observations show a difference in this virus from that of epidemic influenza.

The greatest incidence is during the fall and spring when there are more rapid changes in temperature. The incidence is less during extremely cold weather, and it is possible that a major factor here is that the infected individual is less apt to get out and spread the disease. One of the earliest notes calling attention to the reduced frequency of respiratory diseases in cold weather was from the observations made during Scott's Antarctic Expedition in 1912.

Many predisposing factors have been mentioned. Among them are chilling, exposure to cold, especially following prolonged warmth; drafts, malnutrition, improper diet, especially deficiency in the vitamins A, B, and C; lowered body resistance from previous diseases; chronic foci of infection in the respiratory tract; continued trauma of the mucous membrane of the respiratory system, as from chemical, physical or allergic agents. Mixed crowds are always a potential source of danger.[1]

PROPHYLAXIS

In prophylaxis the avoidance of exposure to the infection is the first consideration. Especially in periods when the disease is

*Presented at the Second Annual Scientific Session of the Louisiana Academy of General Practice, May 7, 1949, New Orleans.

prevalent, the individual may benefit by not visiting the sick, and by staying away from gatherings. The isolation of patients is of major importace, particularly during the first day or two when infectivity is strongest.[1] Isolation of the patient not only prevents spreading the disease but lessens the chances of his acquiring a cross-infection.

To avoid spreading the disease one of the simplest, easiest, and yet the most neglected precaution, is the avoidance of coughing or sneezing when in the presence of others, or in quarters, the air of which may soon be breathed by others. When coughing or sneezing is necessary the face should be covered with a heavy handkerchief if the patient is out; in the bedroom a towel may be used. In an emergency he may pull the bed covering over his face.

Measures for building up resistance have been advocated through the generations, but their value is still problematic. This refers particularly to outdoor exercises, cold baths, and other so-called "hardening" processes. Such measures were somewhat discredited during the last two wars when such a large percentage of soldiers, living under thoroughly ideal conditions from this standpoint, were not protected.

In the hospital wards the use of germicidal lamps and air-conditioning has seemed to be of considerable benefit.[4]

At one time vitamins were enthusiastically advocated, especially vitamin A, and this product received the synonym of "the anti-infective vitamin". It is highly probable that a vitamin deficiency may render one more susceptible to the disease, but it is not thought today that any excess of vitamins above the normal requirements will lessen susceptibility.

The value of bacterial vaccines is still a moot question. As the common cold is a virus infection it would not seem logical that bacterial vaccines would convey any direct immunity. The period of immunity even from the disease is comparatively short and somewhat imperfect[5]. It is possible that the development of complications from secondary bacterial invasion may be reduced. "The use of vaccines is still considered to be in the experimental stage".[6] Extensive clinical experiments, carried out with adequate controls indicated that vaccines, orally or hypodermically, had no effect.[8] It should be remembered that the A and B influenza virus vaccine carries no protection against the common cold.[7]

Quinine in the treatment of colds was at one time one of the most popular remedies. One recent report, not only claimed good results in treatment, but excellent prophylactic benefit by the use of 8 to 10 grains daily during periods of epidemic. Against the use of this agent might be argued its well known effect on the auditory apparatus, which might add to the burden imposed by the disease itself on those structures.[9] The prophylactic use of penicillin has been reported as having value.[10]

GENERAL CARE

In therapy, proper rest in bed seems to be the only measure that is universally accepted as having a definite influence on the course of the disease.[5] This is not followed as rigidly as is the case in epidemic influenza. Many colds are so mild that any large measure of restriction would seem inadvisable. The degree of rest is best dictated by the amount of discomfort the patient is experiencing and the height of the temperature. In practically all uncomplicated cases the patient may have bathroom privileges. The major considerations are to prevent the patient spreading the disease and to lessen the chances of acquiring a secondary bacterial infection. In any event, exposure to drafts, chilling, and getting wet are best avoided. Except in the presence of a material elevation of temperature or other symptoms, such as sore throat, persistent cough or nausea, little, if any, dietary restrictions are indicated. There may be some advantage in the old custom of emphasizing cool fruit juices and hot milk drinks.

As the respiratory tract is irritated, smoking would seem to be inadvisable. We have a clue here to the protective processes of nature, as many of these patients are robbed of their desire for smoking during the course of the disease.

DRUG THERAPY

It is well to acknowledge in the beginning that we have no specific therapy. On the whole, drugs exert little influence on the course of the infection or on the percentage of complications,[1] and treatment is almost purely symptomatic.[2] Through the ages various drugs and remedial measures have been advocated as having more or less specific value. Many of us can remember the time when it was thought that most colds could be aborted by diaphoresis. An old routine was the hot mustard foot bath, with the patient draped in a blanket that surrounded the foot tub. During this time a large dose of Dover's powder and a large hot drink (with or without a spike) were administered. The patient was then put to bed in a closed room and warmly covered. I sometimes imagine that my mouth still burns from the hot sassafras tea which was the beverage for this regime in my community when I was a boy.

The time honored initial purgative has justly fallen into disrepute. It is probably distinctly contraindicated. "The patient may be kept awake when he should be sleeping; exposed to cold when he should be warm and protected; disturbed when he should be at rest, and his vital resources depleted when they should be conserved."[5] Should there be any special indication, a mild laxative may be administered in the early morning.

Gargling has been largely discontinued. Nose drops and sprays probably do more harm than good. The oil preparations are considered distinctly dangerous. Most of the vasoconstrictor preparations are followed by vasodilation that leaves the patient worse for their use. When it is necessary to open the nose enough for the patient to go to sleep an isotonic glucose solution of ephedrine sometimes may be used to advantage. Of course, in the presence of complications, such as otitis media or sinusitis, sprays may be indicated. Argyrol is at least questionable. The benefit is limited or absent and the chances of argyria are too definite to justify the risk. Quinine is now seldom employed. It still has its advocates.[9] The value of atropine is still an undetermined problem. My own impression is that in small doses it has a field of usefulness where there is a tendency to rhinorrhea. Papaverine, especially with codeine,[11] has been advocated for many years. The present tendency is away from its use.[3] Codeine alone, or with other agents, is probably one of the safest and most efficient remedies for promoting comfort. Coal tar derivatives, particularly aspirin, are almost universally employed. The impression is that aspirin is the most satisfactory and that its value is limited to promoting comfort. Bismuth, especially the intramuscular injections of the salicylate in oil, has been advocated.[12] It is probable that its usefulnes is limited to the prevention or relief of complications from certain types of organisms. Various alkalinizing agents have been largely used and may have some value.

Among the various other agents recently recommended are: propadrin,[13], gamma globulin,[14] ascorbic acid [15] and benadryl.[16] It is probable that the antihistamine drugs have a tendency to lessen the discomfort, especially of rhinitis. In many patients they also act as sedatives.

Last, but not least, are penicillin and the sulfonamides. While good results have been reported from these agents,[17, 18] the impression is that "Sulfonamides and penicillin do not shorten the course of colds and they should be used only when signs of involvement of the sinuses, tonsils, or lungs are apparent."[1] It is held that since most acute respiratory diseases are due to viruses, which are not affected by these agents, the only possible justification for their blanket administration is as a prophylactic measure against complicating bacterial infections.[19]

For about twenty years I had charge of the medical treatment of the student nurses at the Southern Baptist Hospital in New Orleans. These young women were instructed to report promptly to the nursing school authorities upon any evidence of any illness. In the case of colds, if they showed fever they were put to bed and kept there

until the temperature had been normal for twenty-four consecutive hours. They were given sodium bicarbonate, 1 Gram, and sodium citrate, 2 Grams, about every three hours when awake. This was given in carbonated water. Acetylsalicylic acid, 0.3 Gram, when needed for discomfort. Codeine sulphate, 0.03 Gram, when necessary. The patients were instructed not to blow the nose with violence, in order to lessen the tendency to sinusitis or otitis media. They were instructed to cover the face properly when coughing or sneezing. If there was much rhinitis a spray of isotonic dextrose solution of ephedrine was used when necessary to secure normal breathing for sleep. A laxative or enema was used only if there was constipation. In the matter of rest, diet, etc., the cases were managed as outlined in the preceding paragraphs under general care.

In house practice one or both of the following prescriptions are often employed:

℞

Ammon. Brom.	1½ dr. or 6 grams
Sodii Bicarb.	3 dr. or 12 grams
Sodii Citrat.	1 oz. or 30 grams
Liq. Potas. Citrat. q. s. f. oz.	6 or 180 cc.

Sig: Two teaspoonfuls in carbonated water every two or three hours when awake.

℞

Codeinae Sulf.	3 gr. or 0.2 gram
Atropinae Sulf.	1/40 gr. or 0.0015 gram
Camph. Monobrom.	10 gr. or 0.6 gram
Acid Acetylsal	50 gr. or 3.2 grams

M. ft. cap. No. 12

Sig.—One every 4 hours till relieved.

These simple plans are not offered as having any specific value, but our results have been fairly satisfactory.

CONCLUSIONS

In the past there were few conditions for which there were more "cures". We know now that most of these were without value and that some were harmful. Some misconceptions are prevalent today even in the medical profession.

Prophylaxis has made little advance and some suggested measures carry an element of danger.

General care may contribute much to favorable progress and may reduce complications.

We have no specific therapy for the cold as such and effort is directed mainly to promoting comfort and preventing complications. Should secondary infection develop, early recognition and prompt resort to specific treatment may accomplish much—even to saving the patient's life.

REFERENCES

1. Pullen, R. L.: An introductory consideration of respiratory infections, M. Clin. North America 1322 (Nov.) 1947.
2. Rammelkamp, C. H. and Dingle, J. H.: Acute respiratory disease, M. Clin. North America 1368 (Nov.) 1947.
3. Topping, N. H. and Atlas, L. T.: Common cold; note regarding isolation of an agent, Science 106:636 (Dec. 26) 1947.
4. Rosenstern, Iwan: Control of air-borne infection in a nursery for young infants, Am. J. Dis. Child. 75:193 (Feb.) 1948.
5. (a) Bethea, O. W. and Greenlee, R. G.: The treatment of influenza, N. O. M. & S. J. 94:217 (Nov.) 1941.
   (b) Bethea, O. W.: The use of cathartics, J. A. M. A. 107:1298, 1936.
6. Report of Council on Pharmacy and Chemistry and Council on Industrial Health: The use of vaccines for common cold. J. A. M. A. 126:895 (Dec. 2) 1944.
7. Cowan, D. W. and Diehl, H. S.: Cold prevention study; influenza vaccine for prevention of common cold. Minnesota Med. 31:504 (May) 1948.
8. McGee, L. C., Andes, J. E., Plume, C. A. and Hinton, S. H.: "Cold Vaccines" and incidence of common cold. J. A. M. A. 124:555 (Feb. 26) 1944.
9. Feinblatt, H. M.: Role of quinine in colds, influenza and virus pneumonia, New York State J. Med. 45:509 (March 1) 1945.
10. Lapin, J. H.: Prophylaxis of upper respiratory infections in children treated with oral penicillin, J. Pediat. 32:119 (Feb.) 1948.
11. Diehl, H. S.: The common cold, New York State J. Med. 35:109, 1935.
12. Levy, Tracy: Bismuth therapy for colds, J. M. A. A. Georgia 37:172 (May) 1948.
13. Warren, E. W. and Findley, Thomas: Recent advances in pharmacology. M. Clin. North America 417 (March) 1945.
14. Editorial. Gamma globulin vs the common cold. J. A. M. A. 135:293 (May 17) 1947.
15. Brown, W. B., Mahoney, F., Neidringhaus, A., and Locke, A.: Weather and susceptibility in relation to spread of common cold: effect of ascorbic acid in massive dosage on duration. J. Immunol. 50:161 (March) 1945.
16. Brewster, J. M.: Benadryl as therapeutic agent in treatment of common cold. U. S. Nav. Med. Bull. 47810 (Sept.-Oct.) 1947.
17. Faust, F. B. and Simmons, J. M.: Sulfadiazine in treatment of 200 cases of acute catarrhal nasopharyngitis. J. A. M. A. 125:552 (June 24) 1944.
18. Hoffman, W. S., Hofer, J. H. and Gordon, Hyman: Treatment of acute respiratory infection in children with orally administered, unbuffered penicillin solution, J. Pediat. 32:1 (Jan.) 1948
19. Beeson, P. B.: Editorial Comment. Year Book on General Medicine p. 82, 1948.

DISCUSSION

Question: (Dr. J. M. Bodenheimer): Where does the infection stay betwen the epidemics?

Answer: I wish I knew. I have taught so long it does not embarrass me to say I don't know. We think this condition is more or less endemic; endemic almost everywhere. On Scott's expedition to

the Antarctic there were no acute respiratory infection's except one case of pneumonia, and that was in a man who was drunk and lay down in the snow and went to sleep.

Question: (Dr. J. M. Bodenheimer): What is the value of UBA?

Answer: This has not been established in the case of a common cold.

Question: (Dr. M. B. Casteix): What is the value of respiratory vaccines in prevention of complications?

Answer: In a review some time back, I found that no less an authority than Cecil thought they were of distinct value. In the extensive experiments carried out in the mines of South Africa vaccines were reported as useless. The late Dr. Charles Bloom said that vaccines had an efficiency of about 25 to 50 per cent. It is highly possible that these vaccines have a field of usefulness but it is not established at all.

Question: (Dr. O. L. Sanders): Do you recommend aureomycin?

Answer: I have had no experience in this for the common cold. Just in passing I will say that Dr. Paine, who did much of the original work on chloromycetin said it acted so well in so-called virus pneumonias that he refused to be quoted until it was more difinitely established. However, for the simple, common cold I would hesitate to recommend any of these preparations because I believe that most of these infections will be over in a few days. If complications develop it is a different story.

OCULOGLANDULAR TULAREMIA*

J. WILLIAM ROSENTHAL, M. D.
NEW ORLEANS

One often thinks of oculoglandular tularemia as a specific entity. However, as tularemia is studied it becomes apparent that its manifestations are so protean that a total knowledge of the disease is necessary. The ophthalmologist, therefore, attending such a case must be able to diagnose tularemic pneumonia, fluctuating buboes or other manifestations, but should call in one more capable to handle the treatment of them.

EPIDEMIOLOGY

Our knowledge of this disease has steadily grown since McCoy first isolated *Pasturella tularensis* from the livers and spleens of ground squirrels affected during an epizootic in Tulare County, California. (Incidentally, the disease is now uncommon in that region.) The epidemics in man depend upon the epizootics of the vectors. The latter are many: squirrels, rabbits, deer, flies, mosquitoes, ticks, lice, hogs, sheep, house pets, beavers, horse flies, quails, grouse, sage hens, rats, mice, and even fish. Each vector plays a more important role in a different area, and thus the time of human epidemics varies from place to place. In Wisconsin where rabbits cause most infections, and the hunting season is in November, most infections occur during that month. In Arkansas, on the other hand, ticks play the leading role of vector, and most cases are found during the summer months. Occupation plays a role in those being affected. We expect cooks, butchers, hunters, farmers, and laboratory workers, then, to have the disease more than others. Chance infections may occur all year round, and do, as can be seen by the reports in our own locality. (Table 1) Infection can be obtained with or without an abrasion on the hand and in either case may or may not form an ulcer. Squeezing infected ticks can cause the ulceroglandular or oculoglandular type if the eye is rubbed. The typhoid type usually starts from the ingestion of insufficiently cooked, infected meat. The anginal type (ulcer on the tonsil) has been reported due to drinking water frequented by infected insects and to aspiration of infected material by a laboratory worker. Since the incubation period is one to ten days (usually two to five), and because of the speed of modern travel, we may expect to find the disease in any part of the United States or in the world, for that matter, as it is worldwide in its endemic extent.

We should all, therefore, be cognizant of the disease, because its prompt diagnosis and treatment will save the patient many days of morbidity. The mortality used to be from 6 to 10 per cent. With our excellent chemotherapeutic agents now, however, it is 1 per cent or less. Eight hundred and sixty-nine cases have been reported in the United States during the first thirty-six weeks of 1949, with a median of

*Presented at meeting of the Orleans Parish Medical Society, February 13, 1950.

TABLE 1
TULAREMIA CASES*
LOUISIANA
1948

	JANUARY	FEBRUARY	MARCH	APRIL	MAY	JUNE	JULY	AUGUST	SEPTEMBER	OCTOBER	NOVEMBER	DECEMBER
TOTAL	3	7	6	6	5	2	..	..	2	3	2	9
Ascension	..	..	..	1	..	..	..	..	..	..	..	..
Bienville	..	..	..	1	..	..	..	..	..	..	..	..
Bossier	..	..	..	..	1	..	..	..	..	..	..	..
Caddo	..	..	1	1	3	..	..	..	..	2	..	1
DeSoto	..	..	..	..	1	..	..	..	..	..	..	..
E. Baton Rouge	..	..	1	..	..	1	..	..	1	..	1	..
Franklin	..	..	..	..	..	..	..	..	..	..	..	2
Jackson	..	..	1	..	..	..	..	..	..	..	..	..
Lafayette	2	..	..	..	..	..	..	..	..	..	..	..
Lafourche	..	..	1	..	..	..	..	..	..	..	..	..
Madison	..	..	..	..	..	..	..	..	1	..	..	..
Orleans	..	5	..	..	..	..	..	..	..	..	..	3
Ouachita	..	..	..	..	..	..	..	..	..	..	1	..
Rapides	..	..	..	..	..	1	..	..	..	..	..	..
Richland	..	..	..	..	..	..	..	..	..	..	..	2
Sabine	..	..	..	2	..	..	..	..	..	..	..	..
St. Mary	..	..	..	1	..	..	..	..	..	..	..	..
St. Tammany	..	..	..	..	..	..	..	..	..	1	..	..
Terrebonne	..	..	2	..	..	..	..	..	..	..	..	..
Union	..	..	..	..	..	..	..	..	..	..	..	1
Webster	1	2	..	..	..	..	..	..	..	..	..	..

*Louisiana State Department of Health, Tabulation and Analysis Section Dec. 27, 1949

666 cases for the years 1944-1948. This may be compared with a median of 2,886 cases for typhoid and paratyphoid fevers for the same period.

TYPICAL COMPOSITE CASE HISTORY (UNTREATED)

The patient usually presents himself on the second or third day of the disease, with the history of having cleaned a rabbit or found ticks on his body. Two or three days later he develops malaise, with the primary site being sore. The next day he may have a low fever, nausea, sweats, or a chill, and the regional nodes are enlarged and tender. Then the fever rises to 103° to 105° F. with chills and sweats. Regional adenopathy is quite evident and the primary site shows a papule which then ulcerates. If the lesion is on the hand, the epitrochlear and axillary nodes enlarge; if on the leg, the inguinal nodes; if a conjunctival lesion, the preauricular and cervical nodes. In the typhoidal type, vomiting, diarrhea, and abdominal pain develop, plus other general symptoms, no primary lesion being seen. No primary ulcer may occur if the disease is of the glandular type, only adenopathy being found. In the anginal type, the primary ulcer is on the tonsil and the cervical nodes are involved. Multiple primary lesions may be found, both on the exposed and covered parts of the body. Even bilateral conjunctival lesions have been seen. Occasionally, the patient presents himself in the second or third week of the illness, having been treated at home, or ineffectually by another physician. The findings then will be consistent with the stage of the disease.

A bacteremia occurs during the first (or second) week, accounting for such findings as enlargement of the spleen and liver, pneumonia, and meningismus. Therefore, we see that with any primary form of the disease, complications in distant parts of the body may occur.

During the first week, the patient is very ill, with temperatures spiking from a base of 99° to 101° F., up to 103° to 106° F. Headache, backache, nausea, dizziness, and marked prostration are present. The primary ulcer site gets secondarily infected by this time, causing a necrotic punched-out ulcer with sharp edges and a dirty base. Red streaks of angiitis can be seen, causing the buboes to enlarge and to become fluctuant. The total white blood count will average about 12,000 with some increase in polymorphonuclear leucocytes. Skin manifestations are many and varied. They may be: erythematous, vesicular, macular, urticarial, papular, acneform, pustular or maculopapular.

Chest x-ray may reveal an atypical type of bronchopneumonia, often with a small pleural effusion from which organisms may be cultured. The sputum in such cases also contains the organisms. Pericarditis has been found, as well as generalized peritonitis. One group of patients is unable, antigenically, to cope with the disease, and in these, death can occur during the first week. In the typhoidal type, prostration,

abdominal pains, vomiting, and diarrhea can be found.

The second week of the disease usually finds the fever beginning to spike less, but it remains at a high level, gradually descending day after day, so that at the end of the week it is about 101° F. The primary lesion shows signs of healing, with sloughing of the necrotic tissue, and formation of granulation tissue at the end of the week. Lymph glands are usually quite fluctuant. The pneumonic involvement usually spreads, and is evidenced by pleuritic pain, cough, dyspnea, and production of sputum. Meningismus may be manifest. The conjunctival ulcer, which usually is on the upper or lower tarsal conjunctiva, seldom involving the eyeball, follows the course of any other primary ulcer. Occasionally, however, corneal ulcers develop which may go on to perforation. Acute purulent dacryocystitis is a not infrequent complication.

Without treatment, the disease usually lasts two or three months, the fever gradually subsiding, the ulcer healing, the lymph glands rupturing spontaneously, or remaining fluctuant a long time. Pneumonic lesions slowly disappear. Conjunctival lesions heal well, leaving only a slight scar or possible granulation tissue which may have to be touched with silver nitrate. The period of convalescence is long, without proper therapy, lasting months. Mild exacerbations of symptoms often occur, with much fatigue during the remission periods.

METHODS OF DIAGNOSIS

1. *Clinical observation.*

2. *Skin test.* An intradermal skin test with killed *B. tularense* organisms is a good diagnostic procedure during the first week because it is positive in 92 per cent of the cases when agglutinations are still negative. It takes forty-eight hours to get positive. However, it has the disadvantages of all intradermal tests.

3. *Culture.* Organisms apparently may be cultured only on blood-glucose-cystine agar, egg yolk, or other specialized media, which may account for many previous reports of negative cultures. The organisms may be obtained from the sputum, blood, pleural fluid, or the primary lesion, rarely by bubo aspiration. From the culture organisms may be smeared and stained or inoculated into laboratory animals.

4. *Animal inoculation.* Organisms from cultures may be injected into mice or guinea pigs as mentioned above, or any of the sources for culture may be mixed with saline and injected into the animal's peritoneal cavity. The animal usually dies in four to seven days, and on autopsy shows focal necrosis of the liver and spleen which may be cultured as above, or smeared and stained. Because of the danger of laboratory workers contracting the disease even through the unbroken skin, many laboratories refuse to use culture or animal inoculation methods.

5. *Agglutinations.* Aside from the clinical signs, this is probably the most satisfactory method of diagnosis. The test is very reliable. However, it becomes positive only after about two weeks, depending on the case. The first few days of the disease find the titer to be zero. It may be 1:20 on the ninth day of the disease, about 1:80 on the thirteenth, 1:160 on the fifteenth, 1:320 on the seventeenth, and so on, up to about 1:5120 which is about the usual peak, reached in the third week. Levels up to 1:163,840 have been seen. The onset of positive agglutinations also varies widely and was positive only on the forty-third day in one case. After that, the titer gradually falls, but the test remains positive, agglutinins being found throughout life.

The agglutinin level for diagnostic purposes varies with different authors. However, the levels 1:80 and 1:160 are within the "diagnostic range". Levels above these are definitely positive. We must remember, however, several things:

A. Serial agglutinations should be taken, and a rising level is more significant than one which is stationary.

B. Patients who are not treated adequately and may go on to death, may show a zero level, a stationary level, or a decreasing level after an initial high. Some pa-

tients never do develop antibodies, and it is this group that may succumb to the infection in a few days if adequate therapy (streptomycin) is not given from the onset. Therapy here is a lifesaving procedure.

C. In another group of patients streptomycin therapy may be so bacteriocidally efficient that a Herxheimer-like reaction is produced after one or two days' treatment. Excess antigen is present in the circulating blood. This reaction manifests itself as an increase in general symptoms, including malaise, rise in temperature, decrease in agglutination titer, and generally feeling ill again. Local signs (bubo, pneumonia, and ulcer) do not get worse. With continuation of therapy, the patient undergoes rapid recovery.

D. In any severe febrile disease, the existing titer of antigens will rise for any other disease which has antigens in the patient's serum. Thus, a rising titer for typhoid O and H and paratyphoid A and B may occur, initially, with the titer for tularemia. However, the levels for these diseases will not reach diagnostic levels unless the patient has recently recovered from one of the diseases. Also, it will be seen that the level for tularemia, after a few days, will begin to get progressively higher, while the others will stay at a low level.

E. Some human tularemia sera and some artificially prepared antitularense sera agglutinate *Brucella abortus* and *Brucella melitensis*. After an extensive serological search, Francis and Evans[16] concluded that:

"I. Because of the frequent cross agglutination between tularense on the one hand, and for abortus or melitensis on the other, serums from suspected cases of tularemia and undulant fever should be tested for agglutinations of tularense and either abortus or melitensis, unless the clinical history points definitely to a recognized source of infection for tularemia or undulant fever.

"I. A serum which shows a marked difference in titer for tularense on the one hand, and for abortus or melitensis on the other, can usually be classed by the higher titer as due either to tularemia or to one of the varieties of *Brucella melitensis*.

"III. A serum which agglutinates all three organisms to the same or nearly the same titer should be subjected to agglutinin absorption tests."

PATHOLOGY

The pathologist sees two types of lesions—acute and chronic. The acute lesions are seen mostly in the lymph nodes and primary lesion, the spleen, liver and lung showing the changes to a lesser extent. Focal necrosis and suppuration are the characteristic acute findings.

In the chronic forms, the lesions may pathologically be confused with tuberculosis. Central necrosis, with epithelioid and giant cells, is found.

BACTERIOLOGY

Bacterium tularense (called by some, *Pasturella tularensis*) is a small, nonmotile, pleomorphic, Gram negative, aerobic, nonspore forming bacterium. In young cultures, bacillary and ovoid forms predominate, but in old cultures it takes a coccoid form.

The colonies are round, opaque, smooth, and buttery in consistency. It grows only on media containing appreciable amounts of cystine, as egg yolk and blood-glucose-cystine agar.

The organism is killed by:

1. Heat at 56° to 58° C. for ten minutes.
2. Thorough cooking of infected tissue.
3. Formalin (0.1 per cent or 37 per cent formaldehyde) for twenty-four hours if the organism is suspended in saline.

Rabbit tissues kept frozen at -14° C. retain the virulence of the organism for six to eighteen months.

DIFFERENTIAL DIAGNOSIS OF OCULOGLANDULAR TULAREMIA

There are only three diseases which may cause a severe necrotizing conjunctivitis with a preauricular node, plus an acute generalized debility with high fever. These are tularemia, diphtheritic conjunctivitis, and streptococcal conjunctivitis. In all of these, the lids and face are edematous; preauricular, submaxillary, and possibly cervical adenopathy are seen; and the palpebral conjunctiva is red and edematous. A true membrane exists.

Tularemia may be proven early in the disease by: culture of the ulcer or aspirated bubo on blood-glucose-cystine agar, skin test, or animal inoculations. The ulcer edges are sharp and the base is dirty and

necrotic. The true membrane which forms bleeds upon removal. Pus which forms in the conjunctival sac and at the inner canthus is of the usual greenish color due to secondary invaders. Corneal complications are uncommon. Diagnostic methods have already been discussed. Multiple primary sites and a compatible history also help in the diagnosis.

Diphtheritic conjunctivitis of the oculoglandular type, also forms a true membrane and the patient may be acutely ill. There may be a history of diphtheria contact. Here, the membrane is grey, not yellow as in tularemia, and though also removed with difficulty, a hard greyish tissue, in which the vessels are obliterated, is found beneath. The lids in diphtheritic conjunctivitis are stiff and brawny in severe cases, a finding not seen in tularemia. Conjunctival discharge in diphtheria is usually of a glaring yellow color. Corneal complications are frequent. Therapeutic trial of diphtheria antitoxin dropped into the conjunctival sac and given intramuscularly in adequate dosage may be used if still in doubt.

Severe streptococcal conjunctivitis is the third acute condition to be considered. The membrane here spreads very rapidly, and the conjunctival discharge is thin and scanty. The smear and culture should be an early diagnostic help. Corneal involvement occurs early, with necrosis, perforation, and its complications. With such a virulent infection possible, it is wise, therefore, to give local and general penicillin or sulfa therapy, if only as a therapeutic trial, early in the disease.

Since edema of the lids in any of these conditions defies lid eversion, one may consider an orbital cellulitis or abscess in the differential diagnosis, until the conjunctival lesion is seen. Findings in this condition not found in the three above are: proptosis, excess chemosis, tenderness and pain on pressure or rotation of the eyeball, and limitation of ocular motion. X-ray evidence of excess orbital density or sinus infection break-through also may be found.

Other causes of membranous and pseudomembranous conjunctivitis may be considered early, before the general symptoms reach their height. At the onset of fever and prostration, however, the following diseases would be less likely to be present even though they produce an oculoglandular complex: tuberculous conjunctivitis, conjunctivitis due to *B. pseudotuberculosis rodentium*, epidemic keratoconjunctivitis, syphilitic conjunctivitis, and chancre of the lid, Parinaud's conjunctivitis, inclusion blenorrhea, agricultural conjunctivitis, sporotrichosis of the conjunctiva, and conjunctivitis due to the *Micrococcus polymorphicus necroticans* of Pascheff.

According to Duke-Elder,[11] "Each case presenting the clinical picture of a unilateral conjunctivitis with lymphadenopathy should, therefore, be subjected to the following tests:

1. Smears and cultures, which may show sporotrix and leptotrix.
2. Guinea pig inoculation with conjunctival scrapings, which may show tularemia, tuberculosis, or the organism of necrotic infectious conjunctivitis.
3. Partial excision for histological examination which may show leptotrix or tubercles.
4. Blood examination for a Wasserman reaction or an agglutination test for tularemia."

THERAPY

I. Streptomycin. This chemotherapeutic agent is now the proven, tried, and true cure for most cases of tularemia. Within twenty-four to forty-eight hours of administration, the temperature drops precipitously (Figure 1) to near normal levels, all lesions start to heal, and the patient feels about normal, although weak. Two or three grams of streptomycin per patient is on the average the total dose needed, according to Foshay.[15] It should be given intramuscularly in divided doses every three or four hours. I believe 1 gm. of streptomycin, intramuscularly, per day for four to five days, then ½ gm. streptomycin for three to four more days is sufficient for any case of tularemia with or without com-

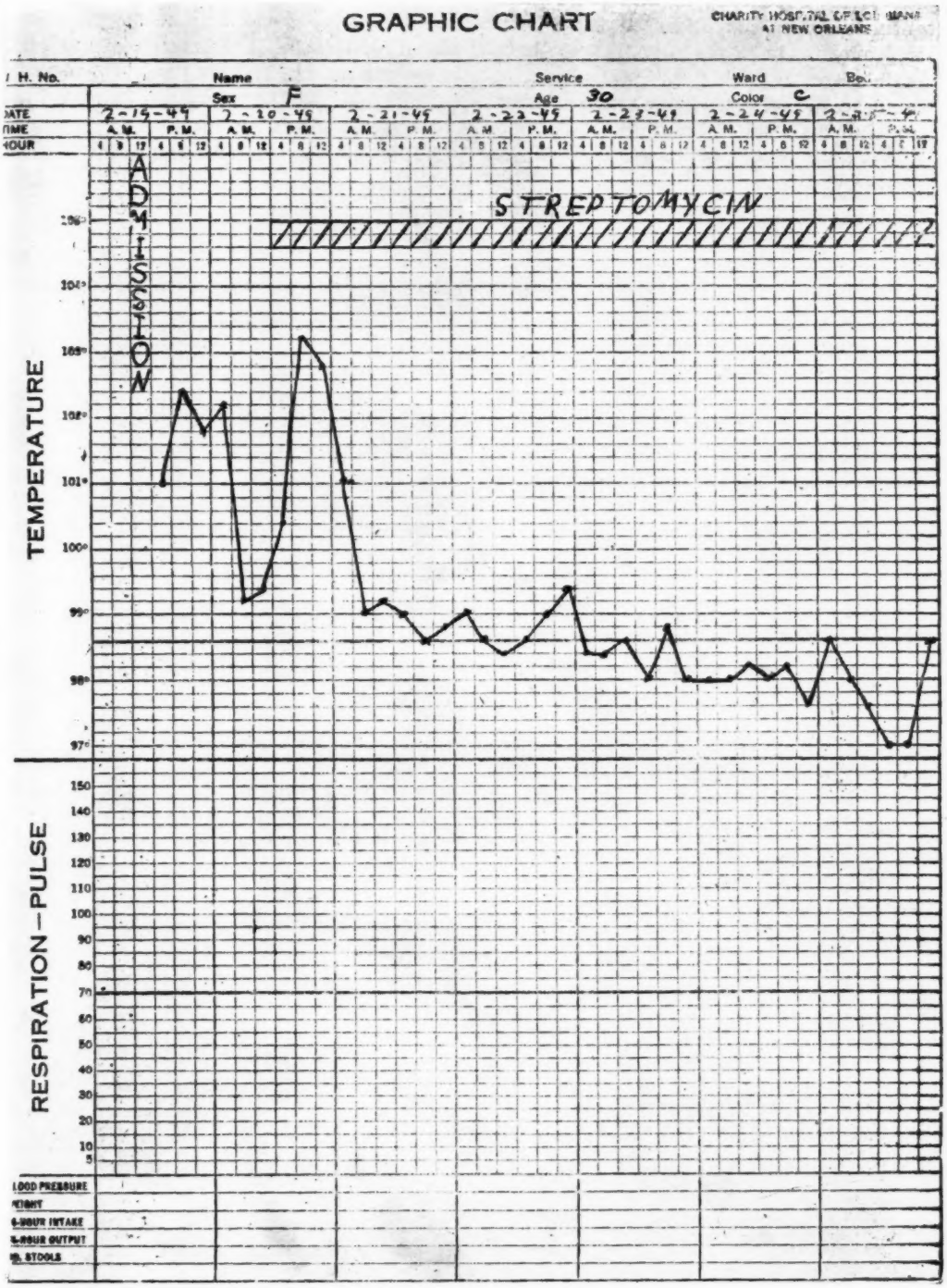
GRAPHIC CHART
CHARITY HOSPITAL OF LOUISIANA AT NEW ORLEANS
Name
Sex F
Service
Age 30
Ward
Color C
2-19-49
2-20-49
2-21-49
2-22-49
2-23-49
2-24-49
ADMISSION
STREPTOMYCIN
TEMPERATURE
RESPIRATION—PULSE
BLOOD PRESSURE
WEIGHT
24-HOUR INTAKE
24-HOUR OUTPUT
NO. STOOLS

Figure 1.

plications. Subcutaneous and intravenous continuous drip may be used to give the streptomycin, but aparently has no therapeutic advantages over the intermittent intramuscular method. (One gram of streptomycin equals one milion units.) Foshay[15] reports that in the blood, streptomycin is bacteriostatic at 0.3 micrograms per cubic centimeter; it is bacteriocidal at 1.0 micrograms per c.c. for one-half hour, and bacteriocidal at 2.0 micrograms per c.c. for eight minutes.

It should be evident from this, that too much streptomycin is usually given to tularemia patients. Some reports in the literature show cures with as little as 1.5 gm. total dose. Prolonging therapy will not heal primary ulcers faster, will not cause buboes to disappear without incision and drainage, will not hasten resolving of pulmonic lesions, and will not make conjunctival ulcers heal faster. However, if the streptomycin is given before a primary lesion (ulcer) forms—that is, when it is in the papule stage,—it will cause regression of the lesion and will prevent its ulceration. This is important, because secondary infection of the primary lesion is prevented which in turn prevents secondary infection of the buboes, which therefore heal faster, and without the necessity for their incision and drainage.

Streptomycin therapy will not prevent development of diagnostic agglutination titers or their proper time of development.

No severe cases of streptomycin toxicity have been reported during tularemia therapy even though 40 grams and over have been given to some patients. Only occasional skin rashes have been seen. Apparently, however, specific and/or regular tests for toxicity have not been made.

Streptomycin is not the total therapy:

1. It may be started too late in the disease, but even though started late, it shortens convalescence materially. However, many of the complications have already occurred, and only time plus natural healing processes can overcome them. Streptomycin, therefore can be expected to be less effective when used late in the disease (third week or more). (Figure 2.)

2. Lack of supportive treatment decreases the patient's feeling of well-being. Sedation is necessary at times; analgesics and antipyretics are certainly in order. Local cleansing therapy to primary ulcers decreases the secondary infection. Certainly the face swelling in the oculoglandular type should be reduced with cold compresses. Here also, the conjunctival sac should be kept clean of exudates by irrigations; ointments should be used to keep the lids from sticking together, and the cornea should be protected by 5 per cent sulfathiazole or penicillin ointments. Patients with pulmonary lesions should have indicated supportive treatment, including pleural cavity aspiration, if necessary.

3. Only a few cases of apparent resistance of the organism to streptomycin have been reported. Most of these have been overcome by increasing the dose of streptomycin. One case was cured by using

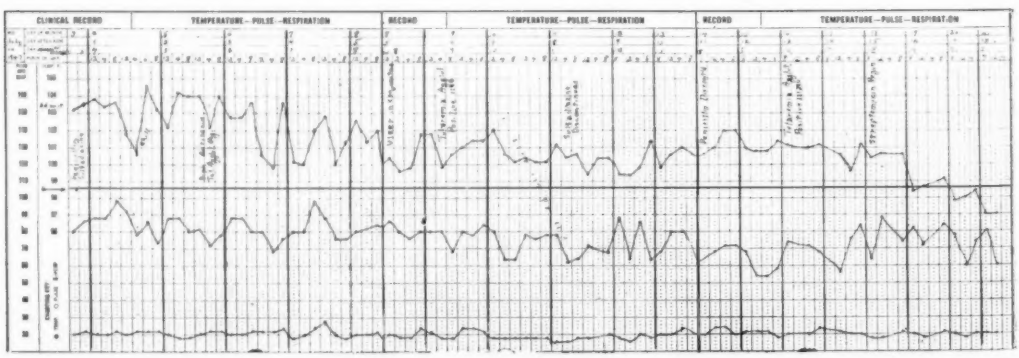

Figure 2.

aureomycin along with streptomycin. These cases have been scattered and inconstant.

Streptomycin, therefore, should be given on the suspicion (clinical or laboratory) of tularemia because (1) it decreases the degree and length of morbidity to less than half of that previously recorded without the drug; (2) it decreases the mortality rate; and (3) it saves the lives of those few patients who do not respond to the infection with adequate white blood cell and antigenic levels.

II. Supportive therapy has already been mentioned.

III. Incision and drainage of buboes is important when they are fluctuant, as they resorb very slowly without it. If they are allowed to rupture spontaneously, which is uncommon, more scarring than necessary is produced, and convalescence is prolonged. Repeated aspiration with injection of streptomycin or penicillin is ineffective.

IV. Aureomycin has been used in mice tularemia, and has been shown to be even more effective than streptomycin. In the few patients where it has been used in man, it has thoroughly lived up to its experimental expectations. It may some day become the drug of choice in tularemia.

V. Chloromycetin as used in mice tularemia was shown to be less effective than streptomycin, but may later prove to be worthwhile if resistant strains of the organism develop.

VI. Polymixin, even experimentally, has not been effective in tularemic infections.

VII. Penicillin and the sulfa group of drugs, although not effective against *Pasturella tularensis*, have their place in the therapy of this disease by combating secondary invaders of the primary ulcers, the buboes, the lung, and the conjunctival sac.

VIII. Foshay's immune and hyperimmune sera, although exceedingly helpful therapy before the advent of streptomycin, have been made obsolete by the latter drug.

IX. The use of convalescent serum is also obsolete.

X. Ecke[12] reports excellent results using 1.5 gr. of atabrine, three times a day for five days, in several unproven cases of tularemia.

SUMMARY AND CONCLUSIONS

1. Because of the diffuse manifestations of tularemia, one must know how to treat any form of the disease, even though as an ophthalmologist one is confronted with an oculoglandular syndrome. An ophthalmologist should, therefore, not treat the oculoglandular type without adequate medical consultation, and conversely, the ophthalmologist should be consulted regarding the eye condition in those cases seen by others.

2. Suppurating lymph nodes and slowly resolving pneumonia account for most of the morbidity time after adequate streptomycin therapy. When fluctuant, the nodes should be incised and drained, only. Resolving pneumonitis should be followed with serial x-rays. Neither of these should prolong hospitalization.

3. Now that streptomycin is available, one should start treatment on the suspicion of the disease rather than wait for laboratory confirmation.

REFERENCES

1. Adler, F. H.: Gifford's Textbook of Ophthalmology, 4th Edition, Philadelphia, 1947.
2. Berson, R. C., and Harwell, A. B.: Am. J. M. Sc. 215:243 (March) 1948.
3. Bost, R. B., Percefull, S. C., Leming, H. E.: J. A. M. A. 137:352 (May 22) 1948.
4. Boyd, Wm.: Textbook of Pathology, 4th Edition, Philadelphia, 1943.
5. Carroll, H. H., and Gorman, J. E.: U. S. Nav. M. Bull., 49:1085 (December) 1949.
6. Chapman, S. S., and Downs, C. M.: J. Infect. Dis. 85:25, July) 1949.
7. Chapman, S. S., *et al*: J. Infect. Dis. 85:39, (July) 1949.
8. Chapman, S. S., *et al*: J. Infect. Dis. 85:45, (July) 1949.
9. Cooper, H. R.: U. S. Nav. M. Bull. 48:102, (Jan.-Feb.) 1948.
10. Craddock, G. B. and Crowder, R. U., Jr.: Virginia M. Monthly, 76:103, (March) 1949.
11. Duke-Elder, W. S.: Textbook of Ophthalmology, Vol. II, p. 1630, St. Louis, 1938.
12. Ecke, D. H. and Ecke, D. H.: Illinois M. J. 93:318 (June) 1948.
13. Fondren, J. T. and Cameron, C. M.: Am. Pract. 3:321, (Jan.) 1949.
14. Foshay, L.: J. Indiana M. A. 41:207, (Feb.) 1948.
15. Foshay, L.: Postgrad. Med. 4:315, (Oct.) 1948.
16. Francis, E. and Evans, A.: Pub. Health Rep. 41:1273, 1926.
17. Francis, E.: Tr. Ass. Am. 60:181, 1947.
18. Glass, G. B. J.: Am. J. M. Sc. 216:411, (Oct.) 1948.
19. Hellman, D. H., *et al*: Am. J. M. Sc. 210:576 1945.
20. Hitch, J. M. and Smith, D. C.: Virginia M. Monthly 65:452, (Aug.) 1938.

21. Johnson, J. B., *et al*: Am. J. M. Sci., 214:645, (Dec.) 1947.
22. Jordan, R. A. and Downs, C. M.: J. Kansas M. Soc., 50:390, (Aug.) 1949.
23. Kilbury, M. J. and Fulmer, S. C., Am. J. Clin. Path. 8:436, (July) 1938.
24. Larson, C. L., *et al*: Ann. New York Acad. Sc., 51:982, (June) 1949.
25. Larson, C. L.: J. Immunol. 62:425, (Aug.) 1949.
26. Lesser, R. E. and Miller, S.: New England J. Med. 238:554, (April 15) 1948.
27. Lindeke, H. I. and Maiden, S. D.: J. A. M. A. 142:99, (Jan. 14) 1950.
28. McCoy, G. W. and Chapin, C. W.: J. Infect. Dis., 10:61, 1912.
29. Morgan, B. B.: Wisconsin M. J., 48:508, (June) 1949.
30. Pasternack, J. G.: J. A. M. A. 112:1814, (May 6) 1939.
31. Public Health Reports, 64:1239, (Sept. 30) 1949.
32. Pullen R. L. and Stuart, B. M.: J. A. M. A. 129:495, 1945.
33. Rigdon, R. H. and Chesnutt, C. R., Jr.: J. Arkansis M. Soc., 44:183, (Jan.) 1948.
34. Ridgon, R. H.: Texas State J. Med., 44:392, (Sept.) 1948.
35. Rosson, R. W.: California Med., 68:137, (March) 1948.
36. Siniscal, A. A.: Am. J. Ophth., 29:698, (June) 1946.
37. Stuart, B. M. and Pullen, R. L.: Am. J. M. Sc., 210:223, 1945.
38. Tassman, I. S.: Eye Manifestations of Internal Diseases, 2nd Edition, St. Louis, 1946.
39. Theobold, G. B.: Am. J. Ophth., 15:112, 1932.
40. Vail, D. T., Sr. :Ophth. Rec., 23:487, 1914.
41. Vail, D. T., Jr.: Arch. Ophth. 55:236, 1926.
42. Vail. D. T., Jr.: Arch. Ophth. 2:416, 1929.
43. Washburn, A. M. and Tuohy, J. H.: South. M. J. 42:60, (Jan.) 1949.
44. Wilson, J. R., Jr.: J. S. Carolina M. A., 45:107, (April) 1949.
45. Zinsser, H. and Bayne-Jones, S.: Textbook of Bacteriology, 8th Ed., New York, 1939.

THE VALUE OF A HEARING CLINIC

PRESENT POLICIES AND LONG-TERM OBJECTIVES OF THE HEARING CLINIC AT THE EYE, EAR, NOSE AND THROAT HOSPITAL IN NEW ORLEANS

LUCIAN W. ALEXANDER, M. D.
NEW ORLEANS

The magnitude of the problem of deafness in the United States is not known. Estimates range from 6,000,000 to 15,000,000 for all types of hearing difficulties. The most exact figures now available are derived from the National Health Survey in 1935-1936,[1] which covered approximately 2,500,000 persons, 48 per cent of whom were males. According to this survey, 1 of every 78 males surveyed, and 1 of every 85 females, had some type of hearing impairment. Between the ages of 35 and 44, 1 of every 103 males and 1 of every 108 females had some hearing loss, the incidence rising with each succeeding decade until between the ages of 65 and 74 years, the distribution was 1 of every 14 males and 1 of every 18 females. Lederer and Hardy[2] estimated in 1946 that there were 20,000 deafened persons in each million of the population and that from 2 to 5 per cent of the school population was hard of hearing.

The implications of these figures are perhaps even more significant than are the statistics themselves. The estimate of deafness in the school population means that from 2 to 5 per cent of all children face life with a handicap which may later make them charges on the state. The estimate that there are 20,000 hard of hearing persons in every million of the population means that no city of any considerable size can escape this special problem. Although the proportion of deafened persons in advanced years reported in 1935-1936 may have remained constant, the actual number of such persons must be considerably larger now than it was then because of the increase in longevity which has occurred over the past ten to fifteen years. Moreover, the concentration of hard of hearing adults in the lower income and unemployed groups which was shown by the National Health Survey[1] clearly indicates that deafness is an occupational and public liability as well as a personal and social handicap.

Otologists in the past have not accepted their full responsibilities in respect to deafness. From the standpoint of active therapy auditory disability has been a discouraging field. As a result, most of us have been inclined to examine the patient, test his hearing, advise him to get a hearing aid, and pay little if any attention to what happened to him after that.

There is no longer any excuse for that line of conduct. The strictly therapeutic aspect of deafness is still not very encouraging, though it has its brighter sides. Certain types of deafness can be cured and others can be improved by therapeutic measures. Certain types can be prevented by appropriate prophylactic measures, the problem here being one of case-finding.

Presented at the Sixty-ninth Annual Meeting of the Louisiana State Medical Society, May 7, 1949.

Finally, types of deafness which are incurable can be managed by a program of rehabilitation. These are incontrovertible facts. It is incumbent upon all otologists to accept them and to act in accordance with them, the best way of translating them into practice being by way of hearing clinics.

Clinics for the deafened and hard of hearing are not new. The Hearing and Speech Center at Johns Hopkins Hospital opened only two years ago, but Crowe's work at that institution, of which it is the culmination, began more than thirty-five years ago. The Otological Research Laboratory at the Abington Memorial Hospital in Philadelphia, founded by the late Dr. Walter Hughson, has been for many years a brilliant illustration of what can be accomplished in this field under the proper setup. It remained, however, for hearing clinics to receive their real impetus during World War II, when brilliant results were accomplished at the Aural Rehabilitation Centers set up at Hoff, Borden, and Deshon General Hospitals under the supervision of Major Leslie E. Morrissett, who served as Consultant in this field in the Office of the Surgeon General. Each of these centers demonstrated what coordinated management can accomplish both in combat-incurred deafness and in the civilian varieties; it must not be forgotten that many of the men who were treated had their disability before they were inducted into service. Since the war numerous hearing clinics have been set up in various parts of the country and for almost the first time there is now becoming available to civilians the same type of expert care which service men received during the war and which is now provided for veterans.

There are in Louisiana about 25,000 deaf persons who are conscious of their defective hearing. Unquestionably, the number would be much larger if there could be included in it the many persons who have hearing defects of which they are not conscious. Up to last year there were in Louisiana only two institutions devoted to the care of deafened and dumb persons—the institution at Chinchuba, operated by the Roman Catholic Church, and the institution at Scotland, operated by the state. Both are crowded beyond their capacity and both have long waiting lists.

GENERAL POLICIES OF THE HEARING CLINIC AT THE EYE, EAR, NOSE AND THROAT HOSPITAL

This is the story of the first ten months of operation of the Hearing Clinic, which was established at the Eye, Ear, Nose and Throat Hospital in New Orleans, June 1, 1948.

The clinic is not a private enterprise. It is a function of the Eye, Ear, Nose and Throat Hospital. All supplies are provided by the hospital. Space and equipment are provided by the hospital. Clerical and technical services are provided by the hospital. The services of the staff are given without charge, and the cooperation of every local otologist, whether or not he is a member of the hospital staff, is invited and would be welcomed.

All patients who pass through the Hearing Clinic undergo the same routine of investigation and management, regardless of their financial status. Patients who are unable to pay anything pay nothing. Patients who can pay something pay whatever they can afford. Private patients who are financially able pay a maximum of twenty-five dollars for the investigation. All funds received go into the Hearing Clinic and help to finance it, though the fees received do not, of course, come near to meeting the expenses.

Case-Finding.—The Hearing clinic secures patients in several ways:

1. Case-finding is a constant function of the free clinic operated by the hospital. Here the hearing of all persons is tested as part of the clinic routine and those who show any defects are diverted to the Hearing Clinic.

2. Some patients are referred by private physicians, who may lack the time and facilities for diagnostic testing. It is the policy of the Hearing Clinic to treat no patients referred to it by private physicians unless it is clearly instructed to do so. All other patients are returned to the referring

physicians after the work-up.

3. The Hearing Clinic works in close cooperation with the Orleans Parish Public School System, which for many years has had special teachers for the deaf and special classes for deafened children. Continuous case-finding is carried on in the schools, under the direction of a special investigator. Children, who because of inattention or for other reasons are suspected of hearing difficulties, are referred to her, and if the suspicion of deafness is confirmed, they are brought to the Hearing Clinic for full investigation and treatment. Before this facility was established they were merely trained in lip-reading and speech.

4. The Hearing Clinic also cooperates with the Louisiana Employment Service, again with mutual advantage. Persons who have lost their positions because of deafness, or who are unable to find employment for this reason, are investigated in the clinic, and are submitted to the necessary therapy or training. The Employment Service, on the recommendation of the clinic, will furnish hearing aids to persons who from the employability standpoint might be benefited by them.

5. Finally, the Hearing Clinic works in close cooperation with the local branch of the American Hearing Society, again with great mutual benefit. With the establishment of the Hearing Clinic its own work is enhanced. The trained worker assigned to the Clinic by the Society, has taken over many responsibilities which must be met if the full possibilities of such a clinic are to be fulfilled but which are not precisely the work of otologists, who, for that matter, are seldom trained to meet them.

ROUTINE OF INVESTIGATION

The routine of investigation in the Hearing Clinic is about as follows:

1. A complete history is taken and a complete examination made, exactly as they would be carried out in any other general investigation. This and other phases of the work-up are conducted by the internes and residents (six of each) attached to the hospital staff.

2. A complete auditory history is taken. Special attention is paid to the family history of hearing and to the possible influence of physiologic function, environment, and other factors on the hearing disability.

3. A complete examination is made of the nose, throat, and ears.

4. Complete hearing tests are carried out, including the whispered and conversational voice tests and the Rinne, Schwabach and Weber tests.

At this point I wish to emphasize the usefulness and the simplicity of tuning fork tests. The equipment is inexpensive and testing can be carried out by any physician. Most general practitioners, in fact, have the tuning forks in their offices and simply fail to use them. If they were universally used, case-finding in deafness would promptly advance by leaps and bounds. Physicians who could not themselves handle the deafened patients thus identified could refer them to otologists who could, or to a hearing clinic, for investigation, evaluation, and treatment or rehabilitation. In my own opinion, one of the most important things any hearing clinic can do is to disseminate information concerning this simple method of case-finding.

5. Audiometric testing. In general, the results of audiometric and tuning fork tests should agree. If they do not, the tests should be repeated until the discrepancy is clarified.

6. Thereafter the investigation is conducted according to the necessities of the special case. In other words, the program is rigid up to this point but is flexible thereafter. Roentgenologic examinations of the head are made in almost all cases. If the general examination of the mouth and teeth has shown the need for special dental consultation, the dentist is called into the case. The staff allergist sees all patients in whom allergy is suspected as a possible background for the hearing defect. Phychologic and psychiatric examinations are conducted as necessary. The evaluation of emotional, attitudinal, and other factors which might militate against rehabilitation is important, but we are extremely careful

not to create psychiatric and psychologic problems where they do not already exist.

7. When all the elements of the investigation have been completed, each case is evaluated by the entire staff. Our practice is to hold a weekly conference, lasting from an hour to an hour and a half, which is attended by all members of the staff and all consultants who can arrange to be present, and at which we are delighted to have the attendance of other interested physicians. From two to six cases are presented each week. The resident who has worked up the case makes the presentation and answers questions by those who have not already seen the patient, or he is examined by them, as fresh points of view are developed. Finally, the management appropriate to his case is outlined.

It is at these conferences that the full value of the coordinated investigation and treatment which are possible in a hearing clinic are most evident. The preliminary work-up is always thorough, or so it seems, until a question or suggestion from some member of the staff who is not familiar with the case opens new avenues of investigation and evaluation. The advantages of a joint approach to therapy and rehabilitation are too obvious to need discusion. Attendance at these conferences is an interesting and stimulating experience and the conferences themselves assure for the patient a well rounded approach to his special needs.

THERAPY

The management of the deafened person consists, according to the necessities of the special case, of medical or surgical therapy, dental treatment, radiotherapy, or narcosynthesis; instruction in lip reading; training in residual hearing; voice training and speech correction; the provision of a hearing aid and training in its proper use; vocational guidance; and any other special measures the individual patient may need to restore him to a useful, profitable existence. The mere listing of these various measures of therapy and rehabilitation makes clear the value of the liaison which has been established with the Public School system, the State Employment Service, and the American Hearing Society.

One or two special forms of therapy might be briefly commented on.

Surgical Measures.—Surgery has a very limited field in the treatment of deafness. We make it a practice to clear up conditions which would need correction even in the absence of deafness, such as infected tonsils, and mastoid and sinus infections, but we resort to surgery only when it is clear that nonsurgical measures are not effective, and in our opinion there is usually little or no relationship between the deafness and the pathologic process. Sometimes an exceedingly simple procedure, such as the removal of an accumulation of cerumen, brings about a surprising degree of improvement in the patient's hearing.

The single surgical measure designed to relieve deafness *per se* is the fenestration operation, which is now generally performed by the nov-ovalis technic of Lempert or by the Shambaugh modification of the Lempert technic. This operation unquestionably represents the most important advance ever made in the active therapy of deafness. Its application, however, is extremely limited. It is useful only in deafness due to clinical otosclerosis, and is definitely contraindicated when nerve damage of any considerable degree is associated. It is sometimes useful in certain borderline cases of cochlear nerve degeneration, though there is at present no accurate, reliable test for estimation of the reserve cochlear nerve function. When the operation is indicated, when it is limited to cases in which it is suitable, and when the surgeon has been fully trained in the technic, it is sometimes brilliantly successful.

The number of cases in which the fenestration operation is indicated is, as has been intimated, very small. The field of all surgical procedures is, for that matter, very limited. Of the 105 patients who have passed through the Hearing Clinic at the Eye, Ear, Nose and Throat Hospital during the ten months of its operation, only

21, (20 per cent), were regarded as suitable candidates for any sort of surgery.*

Dental Measures.—It may come as a surprise to many physicians to hear that the dentist attached to the staff of the Hearing Clinic is one of its busiest and most useful members. It is unfortunate that there is no general understanding of the possible relationship between dental defects and hearing disabilities. Costen[3] of St. Louis, and Goodfriend[4] of Philadelphia were the first to point out this relationship and to emphasize the etiologic importance of malocclusion which, according to Goodfriend, is a possible factor in 40 per cent of all cases of deafness. Furthermore, while it may be merely coincidental, it is nonetheless striking to find how closely the incidence of deafness parallels the incidence of edentulousness in the various age groups.[5]

When there has been a loss of teeth, particularly the molars, and there has been no replacement, or replacement has been inadequate, certain anatomic relationships become altered. The intermaxillary distance is decreased, or as dentists prefer to express it, there is a loss of vertical height. The condyles assume abnormal positions when the mandible is closed, being pushed posteriorly, superiorly, or medially. Sometimes there is actual erosion of the bony portion of the eustachian tube, with subsequent blockage by the mandibular condyle. Sometimes there is compression of the membranous portion of the eustachian tube by the shortened external pterygoid muscle. Sometimes tubular ventilation is decreased because of shortening of the *tensor veli palatini* muscle. Sometimes there is reflex contraction of the *tensor tympani* muscle, as the result of long-continued irritation by the condyle, with resulting loss of function of the tympanic membrane in the conduction apparatus.

Even when missing teeth have been replaced by artificial dentures it cannot be taken for granted that there has been no loss of vertical height. It is possible that the dentures were not constructed properly in the first place. While in all cases, since alveolar resorption is a continuous—albeit individual—process, it is quite possible that dentures which originally fitted well no longer fit as they should.

Deafness due to dental defects is at first conductive. Later, if it is not corrected, secondary nerve degeneration occurs also. Fortunately, if the etiologic factors are recognized promptly, deafness of this origin is reversible: reconstitution of the normal vertical dimensions in turn corrects the abnormalities of the mandibular condyles. We have in our files records of gains of as much as 25 db. in patients who have had no treatment except correction of the bite.

Radiotherapy.—Crowe's work with radon in deafness caused by lymphoid hyperplasia is another form of treatment which received mass confirmation in World War II. Let me briefly review it. His audiometric and other studies on children showed (1) that a surprising number without visible evidence of deafness, or with only slight evidence, had lost their hearing for high tones (above high C), while retaining their hearing for low tones; (2) that children subject to frequent colds were most likely to show this type of auditory impairment; (3) that their hearing disability could be traced to the presence of lymphoid tissue which blocked the eustachian tubes and interfered with middle ear ventilation; (4) that even when adenoid tissue had been removed at operation, it sometimes grew back and produced these effects; (5) that if the condition was not corrected, one octave after another might be affected, so that eventually half or more of the hearing in both ears might be lost, even for the middle range of spoken tones.

For this type of deafness Crowe figured out a remedy, the application of radon. If treatment were instituted early enough, the hearing loss could be completely reversed; while even if it were instituted late, there

*Although we would never perform a fenestration operation on the mere indication of tinnitus, we have observed in a number of cases that this extremely annoying symptom has been greatly improved, or entirely eliminated, following this operation. We have also treated tinnitus by other methods, such as vitamin B therapy, with very good results. The deafened patient's lot is always improved when tinnitus is no longer part of the picture, and auditory acuity is not infrequently increased as a result.

were many cases in which the progression of the loss could be checked.

The Air Force in World War II was suffering a heavy loss of man power from aerotitis caused by lymphoid hyperplasia when it was realized that the principles of treatment devised by Crowe were entirely applicable to this situation. The next step was the development of an applicator to contain radium element (50 gm.) and suitable for use in the limited space in which the application had to be made; radon, of course, was quite impractical for military needs. The final step was the examination of all aviators to confirm or exclude the presence of excess lymphoid tissue and the use of radium in cases in which it was present. How many flying personnel were restored to usefulness by this method I suppose will never be known, but those of us in the Hearing Clinic can testify to its usefulness in civilian otology, particularly in children. It might be added, however, that before radium is applied, it is a wise plan to test the child to determine whether lymphoid hyperplasia is on an allergic basis. If it is, therapy directed to that etiology should be given an adequate trial.

Narcosynthesis.—The Army experience in World War II showed clearly the value of narcosynthesis, in the hands of a competent psychologist, in cases of deafness which have arisen on a functional basis or in which there is a functional overlay.[6] The mass trial of this method at the Hoff General Hospital Aural Rehabilitation Center showed that about three-quarters of the carefully selected group of patients who were thus treated had a complete return of hearing or a great improvement in auditory acuity, both clinically and audiometrically, even though in some cases the deafness had been present for ten years and more. The number of patients likely to be benefited by narcosynthesis is small, and its contribution to the major problem of deafness is correspondingly small, but our own limited experience at the Hearing Clinic is in accord with the Army experience with this method: Its proper application in cases in which it is indicated can produce brilliant results.

REHABILITATION

Hearing Aids.—The provision of hearing aids is one of the most important and one of the most controversial activities of any hearing clinic. Berry,[7] in 1947, estimated that there were in the United States some 800,000 persons who used hearing aids, though he would have expressed it more correctly if he had said that this number had purchased hearing aids. Half or more of all persons who buy prostheses never use them, or use them only briefly or transiently, for a variety of reasons, of which the following seem to be the most important:

1. Perhaps the aid was not needed. A person with a loss of 20-40 to 20-45 db. need scarcely go to the trouble and expense of securing an aid. His hearing will be sufficiently improved by training in residual hearing and in the attention factor. A person with a loss of 70 db. or more will derive so little benefit from the use of an aid that his time and energy would be better spent in learning lip-reading. The person with a loss of 40-70 db. represents the type of deafened person who will be greatly benefited by the provision of an aid if it is properly selected and constantly worn, and if he is properly trained in its use.

In this connection it should be pointed out that the occupation of the deafened person should be taken into consideration in determining his need for a hearing aid. A student or a professional or clerical worker needs one. A farmer or a laborer might manage just as well without one. A machinist working in a noisy factory would do better without one. Incidentally, other things being equal, noisy work should not be recommended for a person with defective hearing.

2. Hearing aids are frequently unsatisfactory because they have not been properly selected. Many, if not most, aids are secured in about the same manner that the purchasers would have purchased any other commodity, that is, without proper medical and technical supervision. I have the highest respect for the work that has been done

by the various companies which manufacture hearing aids, and I am in no way impugning their good faith when I say that their salesmen should no more be entrusted with the selection and fitting of hearing aids—which is in effect the prescription of aids—than opticians should be entrusted with the selection of lenses. In fact, there is even less logic in their being entrusted with this task. The eye has an accommodative mechanism, and the provision of appropriate lenses restores vision. The ear has no similar mechanism, and a long-deafened ear simply does not adjust itself immediately to the sudden amplification of sound produced by the application of a hearing aid. It should be the task of the otologist, aided by experienced technicians, to determine the ear to be fitted, to prescribe the proper kind of aid (air conduction or bone conduction), and to settle other matters in connection with the choice of an aid. Many otologists have pointed out that commercial companies would lose nothing, and inded would benefit in the end, if they would devote themselves to the production and constant improvement of hearing aid and would leave the selection and fitting to those qualified for these tasks.

3. A final reason why hearing aids are unsatisfactory has to do with the fitting, or rather the nonfitting, of the ear piece. This is a fact apparently not realized by those otologists who do not insist upon individually fitted ear pieces or by the commercial companies which provide only the stock insert. There is a difference of as much as 20 db. in the accuracy of reception secured with custom built and stock ear molds. Unless the mold fits perfectly, leakage occurs. When leakage occurs, sounds which reach the middle ear are weaker, and to compensate for the weakening, a higher acoustic output must be used. It is well known that the higher the acoustic output, the less is the sensitivity of the instrument and the less satisfaction the user will have with it. The squeal of acoustic feed-back is another result of leakage which is not generally appreciated.

A hearing aid is not properly fitted unless, in the making of the ear insert, possible retraction of the mandibular condyles is taken into consideration. When the condyles are in their normal anatomic location and the function of the mandibular joint is correspondingly normal, movement within the external auditory canal is at a minimum. When the condyles are retracted, movement increases with the degree of retraction. A recollection of these facts will make clear how important it is to determine, in the fitting of the air insert, whether malocclusion exists and retraction of the condyles has followed.

Anderson, Manion and Cota's[8] studies at the Eye, Ear, Nose and Throat Hospital make clear how little attention is generally paid to this consideration. An examination of 48 consecutive hard-of-hearing patients who wore hearing aids showed that malocclusion was present in all but 20 per cent, of such a degree that abnormal movement of the mandibular condyles was easily demonstrable. In some instances the discomfort on eating or coughing was so great that the ear inserts had to be removed during these activities. Yet careful inquiries elicited the fact that in not a single one of the 48 patients had the state of the teeth been taken into consideration in the fitting of the aids. Well over half of the patients who wore aids expressed themselves as dissatisfied with them, and it seems reasonable to assume that the source of at least part of their dissatisfaction was failure to correct malocclusion.

Auditory Training.—Classes in lip reading and voice training form part of the scheduled activities of the Hearing Clinic. Far more important, however, is auditory training, whether it takes the form of conservation of residual hearing, or instruction in the use of a hearing aid, or simple training in the factor of attention. Many persons who are far from totally deaf might as well be because they suffer from what Macfarlan[9] has well termed "auditory listlessness." They do not hear well enough to make it worth their while to listen. They find the effort of listening fatiguing, and as they make fewer attempts to hear, their

hearing becomes more and more impaired, suffering, for all practical purposes, from the atrophy of disuse. Auditory listlessness cannot be precisely charted. It is largely an individual matter, being closely related to the patient's natural perceptive abilities, mental alertness, and previous education and experience. With some persons it may occur when the hearing loss is 20 db. or even less. With others it may not occur until the loss is 30 db. or more.

The conservation of residual hearing is far more useful to the deafened patient than any amount of training in lip reading. Moreover, it preserves the natural ability of the deafened person's own voice, which is an extremely desirable result.

No hearing aid will be as satisfactory as it should be until the wearer is thoroughly trained in its use. The Army experience furnished particularly convincing proof of this fact and Silverman[10] and others have had similarly convincing experiences in civilian practice.

Training in the use of a hearing aid includes the gradual development of tolerance for amplified sound, refinement of discrimination of speech sounds through amplication, social and vocational adjustments, and the actual mechanics of wearing and caring for an aid. It also includes encouraging the patient to wear his aid constantly. As Hughson and Thompson[11] have pointed out, deafened persons who express themselves as unqualifiedly satisfied with their aids will almost always be found to be persons who are wearing their aids at all times.

THE DEAFENED CHILD

I want to comment particularly on the work we are doing with children at the Hearing Clinic. Modern technics require that they be treated as early in life as possible. If their deafness is not identified, and if treatment and training are delayed until they are 5 or 6 years of age, or older, they begin their education with a handicap because they are behind the children of their own age in language usages and appreciation. We have fitted hearing aids on children as young as 3 years, and our results with preschool children form one of the most rewarding phases of our work.

Auditory training is of great value in children who are congenitally deaf. These children, it has been shown, usually have some degree of residual hearing which, if properly fostered and brought up, can become really useful. We have the necessary equipment for this purpose at the Hearing Clinic and we have had some surprisingly good results in this group of children.

Examination of all children from the standpoint of possible lymphoid hyperplasia is another field in which mass prophylaxis would yield brilliant results. A hearing loss of 9 db. is definitely measurable, and a loss of this amount, while it causes no immediate hearing difficulties, is a warning signal. Retesting at intervals is indicated in such cases, while in more advanced cases treatment with radium by the Crowe method, or with appropriate measures for an allergic etiology, can check the pathologic process and will often result in full auditory ability.

It is extremely important that the parents of a deafened child be carefully instructed in the direction of his activities and training. Training the parents, in fact, is one of the most important phases of the child's rehabilitation. Some clinics such as the John Tracy Clinic in Los Angeles will not accept the child without the mother.

Let me give you an illustration of how a child would be treated in the Hearing Clinic. Here is a 7 year old boy whose nerve deafness is quite evidently the result of the induction of labor by quinine in his mother, who was an elderly primipara with impending toxemia. Quinine induction of labor, as you probably know, is the cause of nerve-deafness in a surprising proportion of cases, and a regrettable proportion, since the condition is entirely irreversible. In the truest sense of the words, the child is born deaf.

This boy's deafness was detected in school, when he was found to have language difficulties; his parents had noted them but had apparently not appreciated their significance. He could not say certain sounds,

"s," "th" and "p," for instance, because he had never heard them. He was found to be a surprisingly competent lip-reader, an ability which he had picked up in school, but he was frequently inattentive, as might have been expected, because he simply did not hear enough to keep him interested.

We shall treat this child with nucleoprotein combined with thiamine, the nucleoprotein being a new preparation just now undergoing clinical testing. We know that it cannot reverse the nerve damage, but we hope that it may prevent further degeneration. We shall see to it that the child's ability to read lips is improved in the special classes held for this purpose as part of the work in the public schools, and he will receive auditory training and speech training in other classes; in the brief time he has been under treatment his speech has already improved considerably. We shall fit him with a hearing aid and train him in its use. He will be watched at school, to be sure that he is seated where he can best hear what is going on, and his progress will be checked frequently with his teacher. Finally, his mother has already been carefully instructed in how to direct and manage him at home.

This is a bright, attractive child, and I have no doubt that if he continues under the joint care of the staff of the Hearing Clinic and of the very competent and enthusiastic staff of the Medical Department of the Public School System, he can be guided successfully through school and into a useful occupation afterward. What might have happened to him if his decided hearing disability had not been discovered is so obvious that I need not expatiate on it.

SUMMARY AND CONCLUSIONS

This is, in brief, the story of what the Hearing Clinic at the Eye, Ear, Nose and Throat Hospital has accomplished and is trying to accomplish. At the present time we are operating under the difficulties imposed by the construction under way at the hospital. When the work is finished, we shall have additional space. That will not, however, solve our financial problems. The resources of the hospital are limited and they must, of course, be spread in many directions. The Hearing Clinic cannot expect from the hospital much more than it is now receiving. Yet it cannot function without minimum expensive equipment, particularly electronic equipment, for which there is no acceptable substitute, or without additional personnel.

With additional funds, there is a great deal that we could do. We could expand our research program, which at present is small because of limitations of money, equipment and personnel. It would be highly desirable if we could increase the number of internes and residents now in training. This is a much needed field of usefulness. Lederer[12] is probably correct in his estimate that at the present time less than 1 per cent of all otologists are qualified to handle cases of deafness by the new technics. Finally, at the present time, because of limitations of facilities and personnel, we are able to admit only six patients each week to the Hearing Clinic. We are, I think, doing a good work with the means at hand, but until we can increase our present intake many times over, we shall be making only an infinitesimal contribution to the problem of deafness as it exists in this city and state.

REFERENCES

1. Beasley, Willis C.: The general problem of deafness in the population. Laryngoscope 50:856, 1940.

2. Lederer, Francis L., and Hardy, William G.: Three types of civilian hearing clinics. A program of physical and psychosocial rehabilitation. Arch. Otolaryng. 45:429, 1946.

3. Costen, J. B.: Neuralgias and ear symptoms associated with the disturbed function of the temporamandibular joint. J. A. M. A. 107:252, 1936.

4. Goodfriend, D. J.: Deafness, tinnitus, vertigo, and neuralgia. Arch. Otolaryng. 46:1, 1947.

5. Brekhus, cited by Anderson et al.[8]

6. Bergman, Moe: Narcosynthesis in the management of functional deafness. In press. Morrissett, Leslie E.: The plight of the nerve-deaf patient. The uselessness of all present therapy. The practical usefulness of aural rehabilitation. In press.

7. Berry, Gordon: Hearing aids. The evaluation and practicability of hearing aids, with review of recent literature. Ann. Otol., Rhin. & Laryng. 57:500, 1948.

8. Anderson, Jack R., Manion, William, and Cota, Agnes C.: Dental considerations in fitting hearing aid inserts. In press.

9. MacFarlan, Douglas: Using residual hearing. Reprint (not numbered) from Hearing News, November, 1939.

10. Silverman, S. Richard: Training for optimum use of hearing aids. Laryngoscope 54:29, 1944.

11. Hughson, Walter, and Thompson, Eva: The hearing aid from the patient's point of view. Arch. Otolaryng. 38:252, 1943.

12. Civilian hearing clinics. Reprint No. 161, from Hearing News, July and September, 1946.

DISCUSSION

Dr. Hartwig M. Adler (New Orleans): I agree with Dr. Alexander that you can't do an accurate evaluation of hearing other than by the audiometric method. At the Metairie Country Day School, one of the teachers does a psychometric examination on all the children entering that school. This is more difficult than doing a whispered and spoken voice test. It should be possible to train teachers, who teach reading, or elocution, in the lower grades to do a more or less accurate screening of their pupils' hearing with spoken and whispered voice tests. The apparently hard of hearing ones could then have their hearing more accurately evaluated by audiometer.

Dr. L. W. Alexander (New Orleans): The testing of very young children is one of the biggest problems we have today. There are very few men who have worked out anything very satisfactory at the present time. I have just recently received some tests that have been worked out in Northwestern University for testing pre-school children. Men doing a great deal of this research are finding a great difficulty in making a very accurate test with these children.

The big problem as I see it is the case-finding. Once you have found the case, the case should be sent t oan otologist who has the means at his disposal to really make a diagnosis. Does that answer your question, Dr. Adler?

Dr. J. D. Martin (Baton Rouge): I would like to tell Dr. Adler that I deliberately did not discuss technics of testing, because I feel that is the otologist's prerogative. I think our job is case finding through every method we have available, then when we have found a child that we suspect of having a hearing loss, we should refer that child to the proper authority for competent medical and other examination to determine whether or not our suspicion is correct.

I would like to stress very much lymphoid hyperplasia, as a cause of deafness in children; I would like to stress it particularly because it is extremely common, and it is associated with allergy and upper respiratory infections. It is one of the causes most amenable to adequate treatment early, and one which all of us as general practitioners should recognize and refer to competent medical authority for treatment and follow-up care.

Dr. J. W. McLauren, an otorhinolaryngologist who expected to be here today told me in his experience, and he has been very much interested in audiology, that 90 per cent of the children that he is able to help by treatment, are children who have a lymphoid hyperplasia causing their deafness.

Dr. Alexander (In conclusion): Some other tests will have to be devised. The whispered voice is not accurate; it depends on the quality of the whisper, the loudness of the whisper; it also depends on the person, some whisper very loud, and others whisper very softly. You have so many factors to take into consideration. I think it will be solved in the future.

THE PUBLIC HEALTH ASPECTS OF DECREASED HEARING IN CHILDREN

J. D. MARTIN, M. D.
BATON ROUGE

The hard of hearing child is one of the most pathetic, appealing, and yet neglected problems of modern medicine. Today many children with decreased hearing can be benefited by proper therapy administered early, but unfortunately many of these youngsters never receive proper therapy early enough for benefit. In 1945 Walter Hughson,[1] now dead, said: "The greatest stumbling blocks to adequate therapy are the pediatrician and family practitioner. The reasons are a lack of understanding of the problem and an unwillingness to admit an inability to deal adequately with the situation once it has been demonstrated."

The purpose of this paper is not to deal with the purely medical aspects of decreased hearing in children, but to reaffirm that this is a major public health problem and that it is remedial and partly preventable.

Here at the onset of this discussion I wish to differentiate between the deaf child and the child with decreased hearing. The deaf child is one whose hearing is of no practical value for the purpose of communicating with others without special instruction in order to acquire articulate speech. This child is provided for in special programs, whereas the child with decreased hearing is neglected. The hard of hearing child is one who learned to speak casually and unconsciously from speaking persons around him by hearing them, but whose hearing is not as acute as it should be, however, and therefore, he does not learn to speak properly. Faults may occur in pitch, rhythm, voice quality, articulation, or language, even though the neuromuscular mechanism for speech may be in-

Presented at the Sixty-ninth Annual Meeting of the Louisiana State Medical Society, May 7, 1949.

tact. Every hard of hearing child has an associated speech defect and these speech defects should be our clue to investigate that child's hearing acuity. The main difference, therefore, between the deaf, the hard of hearing, and the normal hearing child is language and the older the child at onset of hearing loss the better the prognosis for good speech.

What constitutes decreased hearing? There is no unanimity of opinion here, but all of us are aware that between the extremes of good hearing and deafness is a transitional area in which the various degrees of decreased hearing shade imperceptibly into one another. Obviously, however, a decrease in hearing which does not affect a child's personality or his ability to perform the duties inherent to his state of life is only of theoretical importance or perhaps prognostic significance. If however, the decrease in hearing is sufficient to affect his personality or impair his adjustment, then it is a real problem for him. The important thing is not the decrease in hearing, but the child's adjustment to his handicap. A child with a severe hearing loss, but good adjustment is no problem, but a child with even a slight decrease in hearing and poor adjustment represents a major psychologic, social, and medical problem.

How extensive is this problem of impaired hearing? Estimates range from 5 per cent[2] to 8-10 per cent[3] of children attending school. For the sake of evaluation on the conservative side the low estimate of 5 per cent is accepted as being nearer accuracy; that means that 5 per cent of the children attending school in the United States, or about 1,500,000 children, and to bring it closer home, 5 per cent of the children attending school in Louisiana, or about 33,000 children, have decreased hearing, and are in need of medical care and special educational assistance.

The major causes of decreased hearing are the infectious and contagious diseases of childhood, especially scarlet fever, meningitis, and measles,[4] malocclusions, pronounced lymphoid tissue growth at the openings of the Eustachian tubes,[5] and repeated upper respiratory infections. The percentage of children with decreased hearing is also much greater in underprivileged areas where conditions of poverty, poor housing, and undernutrition exist.[3] If we keep in mind the etiology of decreased hearing, the danger of severe hearing loss can be avoided in many instances by proper diagnosis at the time of the onset of the precipitating cause. Hearing loss is most remedial in its incipiency; the longer it persists the more apt it is to become fixed. With continued neglect the decrease in hearing increases and the chance of recovery is decreased. If we are to progress in hearing conservation, we must suspect the possibility of decreased hearing resulting from the acute infectious and contagious diseases of childhood, and from infections of ears, nose, and throat, and we must institute preventive treatment early.

The manifestations of a decrease in hearing acuity which should make us realize that further study of the child is necessary are: frequent ear pain; running ears; reporting of hearing noises in the ear; retardation in class work; speech defects; inattention and frequent mistakes in carrying out instructions; habitual failure to respond when questioned; failure in school in those subjects which require oral explanation or discussion either by the teacher or the class, but excellence in those subjects which require handedness skills and less hearing.[6]

Nothing can take the place of a high index of suspicion, a desire to find these handicapped youngsters, and our diagnostic acumen in recognizing them; however, we will be considerably aided in our work if we will organize a good case finding program using all available methods of detection.

The best approach in case finding is recognition by the parent that something is wrong with the child. This approach is easily defeated, however, if the physician to whom the parent turns for advice fails to recognize the significance of the abnormal manifestation. Teacher observa-

tion is also good, but has the same limitation. The teacher should associate: retardation in educational achievement;[7] emotional instability;[7] behavior problems; grade repeaters; inattention; requests for assignments to be repeated; peculiar listening posture; talking in too low a tone of voice; irelevant answers; restlessness and evidence of fatigue; a spotty educational record with poor work in those subjects that require hearing the spoken voice,[3] together with those manifestations mentioned earlier with a child having deficient hearing.

For mass case finding surveys the audiometer is increasing in popularity. There are two types of audiometers: the phonograph-speech audiometer and the pure-tone audiometer. Of the two, the pure-tone audiometer is the most practical and useful. While it is posible to test 40 children at one time with the phonograph-speech audiometer and only 1 at a time with the pure-tone audiometer, Charles E. Kinney[3] of Cleveland is of the opinion that approximately the same number of children can be screened in a given time by either method and he prefers the sweep-check method with the pure-tone audiometer because it eliminates rescreening, particularly of the low intelligence cases, the poor groupwork cases, and the poor or slow writers, and most important, it picks up those children with high frequency losses and these last are not identified by the phonograph-speech audiometer.

At first thought the audiometer would seem to be the answer to our case finding problem, but do not forget that like all mechanical devices, it has its limitations and should be only an adjunct to and not a substitute for diagnostic acumen. As with any other mechanical testing device, it only tells what the condition is at the exact moment that the test is made. It will pick up many cases of temporary decrease in hearing during and following upper respiratory infections, and it will not differentiate between temporary and permanent hearing loss. Also, unless used by a skilled operator, a child who thinks can outsmart it. In spite of its limitations, however, each year finds it more extensively used as a case finding tool and it is doing a good job. An ideal screening program provides that each child is tested annually, but in many school systems this is not possible. In systems where annual audiometric testing is not possible testing should be done as often as possible. In addition, the ideal program provides for an audiometric test for each child who has had a communicable disease six weeks after the child is over the acute illness. This case finding service should be offered if at all possible in all school systems.

Let us not forget either that case finding is not our objective; our goal is complete rehabilitation of the child maladjusted as a result of his decrease in hearing acuity. To attain this objective an adequate folow-up program, medical psychologic, educational, and social, is needed. Our job is to help the child adjust to his handicap and guide him to become a useful, self supporting, happy member of society.

Aside from our love for children and our obligation to protect their health and guide them to becoming socially adjusted, there is an economic reason why every effort should be made to conserve hearing. As a child's hearing loss increases, his ability to keep up scholastically with the average hearing child decreases. Madden[8] states that there is no difference between normal hearing and hard of hearing children as far as attentiveness, obedience, and social attitude is concerned, but the hard of hearing are rated lower in leadership and aggressiveness. Bonnell[9] reports that partially deafened children repeat grades about three times more than all other children and that the deafened child who finished the 8th grade will repeat between two and three times, at a total expense to the taxpayer of between one hundred and two hundred dollars. If we accept the conservative estimate that 5 per cent of school children have decreased hearing, then 50 of every 1000 children have decreased hearing. The cost to the taxpayer of these

children repeating two to three grades before completing the first eight grades is considerable. Bonnell[9] says that as expensive as it may apear to buy audiometers, to make careful periodic hearing tests and to follow up the handicapped children, and to see that proper medical steps are taken, it is even more expensive to handle the repeaters whose impaired hearing may escape notice until it has become severe and chronic. Examinations by means of an audiometer reveal over ten times as many cases of partial deafness as former routine medical examinations uncovered. Bonnell,[9] also, points out two significant economic facts: (1) Medical treatment, lip reading instruction, the use of hearing aids and more favorable seating should cut grade repetition by at least 50 per cent. (2) There is increased adult earning power for those children who are saved by early treatment from becoming severely deafened later in life.

If rehabilitation will do so much for the child and the taxpayer, it is well to know how it is accomplished. When a child is found to have decreased hearing, he should be referred immediately for competent medical and dental examination to determine the cause of his decreased hearing and, if possible, eliminate the cause. If removal of the cause does not restore the child's hearing an evaluation to determine the extent of his hearing loss, its status, and his adjustment to his handicap, should then be done so that a proper program of rehabilitation may be instituted. The evaluation of the child's decrease in hearing and its effect on the child should be the result of study by an aurist, a speech therapist, a clinical psychologist, and an educator trained to work with hard of hearing children.

When the evaluation study is completed, plans are made for active rehabilitation. Rehabilitation of a child involves more than rehabilitation of an adult, but the process is easier because of the greater adaptability of youth. The earlier the decrease in hearing is discovered after onset, and the later in childhood the decrease first becomes noticeable, the better the prognosis for adjustment and good speech. In addition to a knowledge of how much of a decrease in hearing a child has, it is most important to know what he can do with what hearing he has. A mental measurement to determine his mental potentialities is essential in planning the child's program. Van Adestine[11] stresses this and is of the opinion that some children have more hearing than they make use of so as to cover up the slowness of their mental operations and thus evade the responsibility to learn something. Assuming that the child has been properly diagnosed, we next must determine what adjustments are necessary to permit the child to progress normally with his class of normal hearing children. The hard of hearing child has three traits which favor his adjustment.[12] These are: (1) the ability to concentrate because he is not disturbed by talk or other sounds; (2) an intense wish to make good on any job; (3) adaptability.

Following diagnosis the children should be grouped according to the severity of their handicap.[13] Children with minor losses need only be referred for medical examination and periodic follow-up. Children with minor but significant decreases in hearing should be seated closer to the teacher. A child with a severe loss in one ear only should be seated so that the good ear is toward the teacher and the class. A child with decreased hearing in both ears should be seated toward the front and instructed to watch the lips of the teacher. During class discussion this child should be permitted to change his seating as needed in order to follow the discussion. Children who cannot get along by special seating alone should be given lip reading instructions. Children with severe bilateral hearing loss who have difficulty keeping up even using lip reading should be given special education in small classes in the regular school. In these classes hearing aids should be available and instruction should be personalized. This last is most important.

In addition teachers should discipline

themselves to assist these children as much as possible and always be patient with them and understanding of their difficulties. In addition the teacher should always stress clear enunciation on the part of the pupils and should personally practice it.

The number of children needing lip reading instruction represents about 1 per cent of the total school population. Lip reading is not difficult, but like any art, it requires constant practice to maintain proficiency. Lip reading is the art of understanding what a person is saying by watching the movement of his lips and tongue. Lip reading facility enables children with decreased hearing to keep pace with their normal hearing companions and decreases the number of children who have to repeat grades. In addition to assisting a child to recognize what others are saying lip reading develops alertness, concentration and self assurance, and all of these contribute to social adjustment.

A child with a slight decrease in hearing which will progress will need lip reading as badly as another child with a substantial decrease in hearing. How should children be selected for lip reading classes? McNutt[10] says the number of decibels of hearing loss is not the only criterion for determining need of lip reading instruction. The same degree of hearing loss may be a definite handicap to one child and very little to another. Other factors influencing the need are: the type of deafness, the child's school achievement and social adjustment and the otologist's recommendation.

If a hearing aid is indicated, a child is never too young to use one, but the hearing aid must be selected by an audiologist to fit the child's personal needs and it must never be so powerful as to damage his residual hearing. It must also be borne in mind that some types of ear trouble may be aggravated by the use of a hearing aid. To buy any hearing aid is therefore not only unwise, but may be dangerous. A properly fitted, necessary hearing aid in addition to improving the child's hearing acuity assists him in retaining and improving his speech and keeping his voice normal.

Hand in glove with every hearing conservation program, therefore, should be a speech therapy program. Voice training is a real need and every school system should employ one or more trained, competent speech therapists.

Now so far we have talked about recognizing and providing for the child with decreased hearing but we have not yet said anything about a positive prevention program. It is far better to prevent than cure and the field of hearing conservation is a fertile ground for a good preventive program.

Prevention of hearing loss, as with all other preventive medicine programs, is based on professional and parental education. Physicians, parents and children must know what diseases are likely to impair hearing. Physicians must teach parents and children which infectious diseases frequently spread to the ears and they must also instruct them in methods of preventing this spread. Parents should know and understand the importance of competent medical care early in infectious diseases and must be educated to seek medical advice.

A program to educate principals and classroom teachers to good health knowledge and practices should be a part of their basic training. In the schools the health practice should be in accord with accepted medical practices. Teachers and principals should exclude from school children with upper respiratory infections. Classrooms should be properly ventilated and the temperature should be regulated so as to avoid extremes. Teachers should watch for children with damp clothes or wet feet and provide for them until the clothes and shoes are dry.

The job for those interested in hearing conservation is not yet complete even though we have just talked about a preventive program. Our goal is reached only when the hearing impairment is corrected, or if this is not possible, when the child approaching adulthood has had all of the

rehabilitative measures mentioned above and then receives specialized job training and is placed in a job where he is self supporting, has taken his rightful position in society and society has accepted him.

In conclusion, I would like to summarize the high points of the public health aspects of the hard of hearing child. Hearing conservation can not be a spasmodic affair; it must be a long range program based on the realization that prevention through education is better and less costly than correction; that if damage has already resulted that medical research has demonstrated that at least half of the children with early manifestations of decreased hearing can be cured with proper treatment of the causative condition and many others can be helped;[3] that rehabilitation starts as early in life as it is possible to discover the decreased hearing and that it continues through the school years until the handicaped person is completely adjusted to his handicap and is accepted by his normal companions in society on an equal footing.

REFERENCES

1. Hughson, Walter, Mitchell, Nelson: Textbook of Pediatrics, 4th Ed. 1945, Saunders p. 737 (Hughson, Walter, Former Director, Otological Research Laboratory, Abington Memorial Hospital, Former Associate in Otolaryngology, School of Medicine, University of Pennsylvania and Graduate School of Medicine, University of Pennsylvania, now deceased.)

2. Gardner, W. H.: Hearing tests in 37 Iowa schools, The Volta Review, 1937.

3. Sellery, C., Morley, A.: Hearing health program in a city school system, Reprint No. 184, Hearing News, (September) 1947.

4. U. S. Census, The Deaf Mute Population of the U. S. 1920; Department of Commerce, Bureau of the Census, Government Printing Office, Washington 1928, quoted by Pintner, Rudolf; Eisenson, John; and Stanton, Mildred, The Psychology of the Physically Handicapped, Chap. V, F. S. Crofts and Co., 1941, N. Y.

5. Crowe, S. J. and Baylor, J. W.: Prevention of deafness, J. A. M. A. 112:585, (February) 1930 quoted by Steninberg, J. D.; Montgomery, H. D., and Gardner, M. D., Results of the World's Fair Hearing Tests—Acoustics—Bell Telephone System Monograph B 1256, 1940.

6. Gardner, W. H.: Hearing surveys and school adjustments, J. Exceptional Children, 6:55, (Nov.) 1939.

7. Pintner, Rudolf, Eisenson, John, and Stanton, Mildred: The Psychology of the Physically Handicapped Child, Chap. V, F. S. Crofts and Co. 1941, N. Y.

8. Madden, R.: The School Status of the Hard of Hearing Child, T. C. Contributions to Education No. 499, Bur. of Pubs. Teacher's College, Columbia University, N. Y. 1931 quoted by Pintner, Rudolf; Eisenson, John and Stanton, Mildred, The Psychology of the Physically Handicapped Child, Chap. V, F. S. Crofts and Co.

9. Bonnell, Baker: The Muskogee program for elementary school children with deficient hearing, Reprint by Graybar Electric Co.

10. McNutt, Ena G.: Lip reading and hearing aids for school children, Reprint No. 157, Hearing News, (February) 1946.

11. Van Adestine, Gertrude: The proper classification of children with impaired hearing, Volta Review, (November) 1938, Reprint No. 493, The Volta Bureau.

12. Facts About the American Society for the Hard of Hearing, National Headquarters, 1537, 35th St., N. W. Washington, D. C.

13. Gardner, Warren H.: Hearing Conservation Program, School Board Journal, (April) 1940.

DISCUSSION

Dr. Hartwig M. Adler (New Orleans): I'd like to ask Dr. Martin if he has made any attempts to standardize spoken voice and whispered voice tests, which can be used on the children by the teachers, who are instructing them in the schools. Among my experiences in the Army, I spent my last eleven months doing the EENT examination of men, who were being discharged, and asking whether they could hear with spoken and whispered voice test. My experience convinces me that if the teachers in the elementary grades, who are teaching the children how to read and speak, were taught some simple rudiments of whispered and spoken voice tests, they could fairly well screen these children, and decide who can hear and who cannot, in the "play-game" fashion.

Dr. J. D. Martin (In conclusion): We haven't done any of that work in Baton Rouge for two reasons. It has been abandoned where audiometric tests are being done because of the greater ease and accuracy of audiometric testing, and second because in school work verbal or spoken voice tests are unsatisfactory. There is a great deal of noise in the classrooms in schools, and in the hands of untrained persons, such as the ordinary teacher, vocal tests give a most inaccurate idea of the child's ability to hear. There are also other extraneous factors that affect vocal testing. If you had no other method of detecting hearing loss in the child, I would say that vocal testing would be justifiable. I think as an adjunct to other things it could be used.

Probably a little more satisfactory would be just the ordinary ticking of a watch, if you use the same watch on a large number of children and the tick didn't vary in intensity, and you took the normal distance at which the majority hear the tick, I think it would be a little more accurate method. I would believe a more satisfactory means of suspecting hearing loss, however, would be the manifestations that I mentioned earlier, such as inattentiveness, habitual requests to repeat questions, behavior problems, and things of that sort.

NEW ORLEANS
Medical and Surgical Journal
Established 1844
Published by the Louisiana State Medical Society under the jurisdiction of the following named Journal Committee:
E. H. Lawson, M. D., ExOfficio
C. Grenes Cole, M. D., Chairman
E. L. Leckert, M. D., Vice-Chairman
Sam Hobson, M. D., Secretary
C. M. Horton, M. D.
Leon J. Menville, M. D.
EDITORIAL STAFF
Philip H. Jones, M. D.....*Editor*
COLLABORATORS—COUNCILORS
Edwin L. Zander, M. D.
Joseph S. Kopfler, M. D.
Guy R. Jones, M. D.
Paul D. Abramson, M. D.
C. Prentiss Gray, Jr., M. D.
W. E. Barker, Jr., M. D.
J. W. Faulk, M. D.
O. B. Owens, M. D.
Paul T. Talbot, M. D.....*General Manager*
1430 Tulane Avenue

SUBSCRIPTION TERMS: $4.00 per year in advance, postage paid, for the United States; $4.50 per year for all foreign countries belonging to the Postal Union.

News material for publication should be received not later than the eighteenth of the month preceding publication. Orders for reprints must be sent in duplicate when returning galley proof.

Manuscripts should be addressed to the Editor, 1430 Tulane Ave., New Orleans, La.

THE JOURNAL *does not hold itself responsible for statements made by any contributor.*

STATE SOCIETY OFFICERS FOR THE YEAR

The membership of the Society and the House of Delegates are to be congratulated upon the officers selected for the current year.

Our President, Dr. George W. Wright of Monroe, is admirably suited to the duties of an office which are arduous, time consuming, and require an exquisite nicety of judgment. Dr. Wright, as President-elect the past year, has been able to render invaluable service to the Society and to the cause of medicine as a whole. He has also served in the past as president and Councillor of the Fifth District Medical Society. His great experience in the field of medicine as a prominent internist, and his broad contact with the doctors of the whole State endowed him with additional qualities which are going to be most valuable in a year which we all feel will be one in which crucial decisions against state medicine will have to be made. As this is an election year, it is quite clear that the issue of state medicine and Socialism will be before the voters and in the Congress afterwards. It will be the duty of every member to assist Dr. Wright and to cooperate with him in the strenuous efforts this campaign will entail.

Dr. Edwin H. Lawson becomes Past-President, and as such will continue to make available his judgment, experience, and wide talents for the benefit of the Executive Committee.

The President-elect for the year, succeeding Dr. Wright's term, is Dr. Edwin L. Zander. The Society is most fortunate in having Dr. Zander as President-elect at this time. He brings to this position and to the Executive Committee long experience in the Orleans Parish Medical Society as President and in other capacities. In addition, he has served as chairman of the Committee on Congressional Matters for years with great effectiveness and distinction. His contact with the problems of organized medicine has been broad, and his opportunities for service have been many. The membership should feel very happy in this selection.

The First Vice-President is Dr. U. S. Hargrove of Baton Rouge. Dr. Hargrove has been active in the affairs of the East Baton Rouge Medical Society, and to most excellent purpose. The members have great pleasure in thanking him for the conclusion of a most satisfactory and successful convention in which the arrangements were made for the comfort and benefit of all.

The Second Vice-President is Dr. J. Kelly Stone, whose service in the Orleans Parish Medical Society and in the various matters and conflicts that have been assicated with the efforts of the Society to combat state medicine and to promote voluntary insur-

ance brought to the executive council mature judgment and broad experience. He will be a new and valuable member of the Executive Committee.

The Third Vice-President is Dr. Guy Riche of Baton Rouge. Dr. Riche has long been active in the East Baton Rouge Medical Society, and well known there for his maturity of viewpoint and his long experience as a practitioner.

Dr. A. V. Friedrichs was selected as Chairman of the House of Delegates, as he has been since 1944. The members of the House of Delegates admire the smoothness and dispatch with which he assists them in getting through a volume of business which would take many deliberative bodies several days to accomplish. His breadth of vision and impartiality in the administration of that most important office are appreciated by all.

The Vice-Chairman of the House of Delegates is Dr. J. P. Sanders of Shreveport. He was again elected to serve in this capacity, and his counsel and experience in the Executive Committee are highly respected. He has had broad experience as a practitioner, and recently as the result of his leadership in the Academy of General Practice, and as Vice-Chairman of the Rural Health Committee, and his advice will be most useful in the Executive Committee.

Our experienced and resourceful Secretary-Treasurer, Dr. P. T. Talbot, continues in office and will as always provide the Executive Committee, the members, and all inquirers, with excellent, mature, and accurate advice in the matters that affect organized medicine. His unselfishness and sincerity will long be appreciated.

The Council will remain the same as last year, with the exception of Dr. Ashton Thomas of New Orleans, who becomes Councillor for the First District, indicated by the election of Dr. E. L. Zander as President-elect. Dr. Thomas has had wide experience in organized medicine and has served its interests faithfully and for many years in various offices in the Orleans Parish Medical Society and as a member of the Board of Health for the City of New Orleans. He will be a valuable member of the Council.

The coming year promises to be one that will tax the capacities of all the officers and all the members of the State Society. The officers selected will discharge their duties with satisfaction and effectiveness. They will need the full cooperation of the entire membership.

ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

1950 ANNUAL MEETING

The Annual Meeting of the State Society, held in Baton Rouge April 24-26 proved to be one of the most interesting and well attended in the history of the organization. The Committee on Arrangements, composed of members of the East Baton Rouge Parish Medical Society, rendered excellent service in handling preparations for the meeting and all details were taken care of in a very able manner. There were 437 members, 36 guests and 55 exhibitors registered and all agreed that this was an outstanding meeting.

The House of Delegates held two sessions and for information of the members reports of the President, the Committee on Medical Defense and the Committee on Resolutions, with action taken, are printed below. Further data will be carried in subsequent issues of the Journal.

REPORT OF PRESIDENT

Your State Society has devoted the major portion of its efforts during the last year in overcoming numerous attempts made by various pressure groups to socialize, or federalize, the practice of medicine. In doing this these groups introduced a subtle plan to convert the whole of industrial and professional life into a welfare state. Just imagine the great problem that we are now facing with these astute politicians on one side with substantial resources and a great deal of expert legal action to help them put into effect legislation which would destroy not only the great practice of medicine in this great country, but the free enterprise system under which we have grown and prospered. Druggists, nurses, professional men, and women alike would be ruined by such a measure. We must continue to fight those people with alien concepts who seem determined to drive us down the hill to socialism and communism (Reference: John T. Flynn's *The Road Ahead*). We are obtaining good results in our fight. Indeed, it was this Society, not just the officers and committeemen, but also the 1,848 members that helped defeat the President's (and Mr. Ewing's) reorganization Plan No. 1. The work to accomplish this defeat alone received the highest commendation of the American Medical Association, and Louisiana was one of the five states given credit for defeating this piece of socialistic medical enactment. There have been other federal bills involving medical practice, principally HR 6000 which is for the expanding and increasing of benefits to a larger number of clients in the Social Security Agency; *The Social Security Expansion Bill.* HR 1453; *The Federal Aid to Education Bill*, HR 5940; and S. 1411, *Medical Care for School Children*, all of which have been opposed with some success. Most of these bills are now in the hands of committees for release, and some of them are still having hearings.

To muster sufficient opposition against the above measures has required the support of the officers, committees and every member. The various members of committees, the officers, and all the members of the State Society have displayed great loyalty and a desire to aid the State Society in every respect to make their opposition a success. They have also contributed quite a large sum of money for the financial support of the Society for this purpose. For all of this we wish to thank you, and to state that while we are doing everything we can to hasten the day of final success, no one can predict with certainty how soon such a day will arrive. One thing certain is that we have to continue this fight now until we do win!

In June, 1949, the American Medical Association had its meeting in Atlantic City and outlined twenty points for lay sponsored voluntary insurance groups (known as the Co-ops). After careful study some of these principles were found unsound and difficult to enforce. Many of the doctors of the South, Southwest, and West were opposed to some of these principles and found them to be unsound. Therefore, a conference was held in July, 1949, in Little Rock, Arkansas, where the presidents and delegates from Arkansas, Oklahoma, Texas, Mississippi, Kentucky, Tennessee and Louisiana met. Your president and one of your AMA delegates, Dr. J. Q. Graves, attended this meeting. The result of this conference was the formation of a Western and Southwestern group of representatives of the various state societies concerned, whose objectives were in accord, and especially to provide a means to secure a closer understanding by cooperation between the state societies concerned and the AMA, relative to proposed and existing legislation. These representatives met in a conference in Denver, Colorado in August and this Society was represented by the President-elect, Dr. George Wright, and the Secretary-Treasurer, Dr. Paul T. Talbot. The third meeting convened in Oklahoma City, Oklahoma, and the State Society was represented by Dr. George Wright. This meeting was held just prior to the introduction of S. R. 147 and to it may be particularly ascribed success in passage of

this Senate Resolution. There were quite a few procedures necessary to follow in order to have S. R. 147 passed, which was done by an outstanding Senate vote of 60 to 32. A later meeting of these major groups of doctors was held in Denver in February, 1950. This meeting was attended by Dr. George Wright, President-elect, and Dr. P. H. Jones, Editor of the Journal. This meeting was held principally to formulate plans of procedure to oppose HR 1453, "The Social Security Expansion Bill". In February the AMA held a conference in Chicago where the State Society was represented by Drs. Wright, Friedrichs, Zander and Hattaway.

In addition to these conferences representatives from the Congressional Committee have attended meetings and conferences in Washington and Chicago. Officers and delegates have attended meetings of the AMA House of Delegates at the regular and interim sessions. These conferences dealt with current matters of grave importance to the medical profession. Your Society has been represented by members appearing on the programs of various civic clubs and their presentation of the views of the State Medical Society to members of these clubs, the majority of whom are laymen, has informed the public of the interest and constant attention of your State Society to the welfare of the public. The Council on Medical Service and Public Relations has been most energetic in sponsoring and promoting conferences, addresses and other activities in an effort to defeat federalized medicine. Dr. A. V. Friedrichs, a member of this Council, in conjunction with other members in the Parishes of Tangipahoa, Iberville, and Grant have established school health programs as pilot tests in an attempt to follow out the requests of the AMA. The Council has been most active in promotion and extension of voluntary health insurance as offered by the Louisiana Physicians Service. In reference to the latter it will be noted that this company has progressed to the point of needing more room, and also requiring full time services of a director. The part-time secretary employed by the Council has also become a full-time employee of the Louisiana Physicians Service.

The great increase of work as mentioned above has also doubled or quadrupled the amount of work necessary in our executive office, and especially in the office of our Secretary-Treasurer. At the last meeting of the Executive Committee it was unanimously voted to secure an assistant to the Secretary who shall be a physician, and a committee was appointed for this purpose. It is hoped and respectfully urged that the House of Delegates take similar action in order that we may hastily take care of this increased work which is so important to the future of our profession. With an assistant such as this, the Secretary of the State Society could secure closer cooperation between the State Medical Society office and the various component medical societies and with various national and regional conferences. Such an individual could also greatly assist the Council on Medical Service and Public Relations.

During the past year the overlapping of the duties of various committees was particularly noticed and careful observation of the list of committees with their purposes, indicates an over-abundance of committees. This is particularly apparent in the activities of the Rural Health Committee, the Congressional Committee, and the Council on Medical Service and Public Relations. At the present time there is a special committee appointed to review this particular situation and it is hoped that the members of the House of Delegates will give this particular issue careful consideration.

I cannot complete this report without availing myself of this opportunity to express my sincere appreciation to Dr. Talbot and his associates for their advice and knowledge of the society policies. No one, unless having close contact with the office of the State Society, can conceive of the valuable service the Secretary and his assistants are rendering our organization.

Recommendations

1. It is recommended that a licensed physician be employed to act as Assistant Secretary-Treasurer.

2. It is recommended that the number of committees be reduced and the work of those remaining be definitely outlined and coordinated, thus preventing an existing overlapping of purposes.

EDWIN H. LAWSON, M. D.,
President.

Action taken—The following report of the Committee on the President's Report was adopted: The Committee on the President's Report wishes to accept the report as a whole and to approve the two recommendations contained in the report. We wish to commend particularly that part of the report referring to the necessity of continuing the fight against socialized medicine.

—o—

COMMITTEE ON MEDICAL DEFENSE

In October, 1949, two cases were submitted to the Committee on Medical Defense. Both of these were approved for defense and the attorney for the Society advised accordingly. One of these, a threatened suit against a New Orleans physician, is still pending. The other, filed against a doctor of Lafayette, was settled by compromise after approval for compromise was secured from the Committee on Medical Defense and the doctor in question.

Suit has been filed against a New Orleans physician who, while in the employ of a university, handled a case in Jackson, Mississippi, through the Mississippi State Board of Health, which was not terminated to the satisfaction of the patient. The Committee considered defense of this case and came to the conclusion that the member should receive defense; however, it was agreed that such defense should be offered jointly by the State Medical Society and the university in question and the attorney for the State Society is proceeding in this manner.

The Budget and Finance Committee and the Executive Committee have reviewed financial reports of the Medical Defense Fund and these are on file in the office of the Society for review by any member who may wish to see them.

C. B. ERICKSON, M. D.,
Chairman.

Action taken—Report accepted.

—o—

REPORT OF COMMITTEE ON RESOLUTIONS

Baton Rouge Louisiana, House of Delegates,
1950 Annual Meeting
Gentlemen:

On behalf of the House of Delegates and members of the State Society in attendance at the 1950 annual meeting, it is recommended that thanks be expressed to the following individuals and organizations for their participation in this meeting:

Dr. U. S. Hargrove, Chairman of the Committee on Arrangements, for his splendid cooperation in guiding the local committee in preparations for the meeting.

The East Baton Rouge Parish Medical Society, our excellent host for the meeting.

Dr. P. T. Talbot, Secretary of the State Medical Society, for his usual interest and activity in handling details previous to the meeting, although physically handicapped.

To the Secretaries employed by the State Society for their loyal cooperation.

Dr. Donovan C. Browne for his efforts in arranging the scientific program, which was of great interest to all present.

Hon. Senator Russell Long, annual orator, for his valuable and interesting address at the opening session of the meeting.

Hon. William J. Dodd, Lt. Governor of Louisiana, for his words of welcome.

Guests on the scientific program for interesting discussions.

The press and radio stations for space and time devoted to publicity for the meeting.

Secretary of the Louisiana State Board of Medical Examiners for his informative report to the House of Delegates.

Members of the Woman's Auxiliary for their interest in the meeting.

Commercial Companies for providing interesting exhibits in limited space.

Members of the Society who furnished excellent scientific exhibits.

Companies which participated by advertising in the program for the meeting.

Hotel and Motor Court managers for their cooperation in providing best accommodations available for members and guests of the society.

Dr. Roy B. Harrison who served as Secretary in the absence of Dr. Talbot.

Dr. A. V. Friedrichs, Chairman of the House of Delegates, for his efficiency in expediting matters brough to the attention of the House.

To all officers and members of the Society for their attendance and participation in the meeting.

It is recommended that a copy of this report be included in the minutes of this meeting and that a copy be submitted to the New Orleans Medical and Sourgical Journal for publication.

L. O. Clark, M. D., Chairman
N. J. Tessitore, M. D.
T. A. Dekle, M. D.

Action taken — Recommendation that copy of report be included in minutes of meeting of House of delegates and published in the New Orleans Medical and Surgical Journal approved.

LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Third Thursday of every month	Bastrop
Natchitoches	Second Tuesday of every month	
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

DR. GELBKE HONORED ON 50th YEAR

Colleagues of the Second Medical District last night paid tribute to Dr. Charles F. Gelbke for 50 years of service to citizens of the West Bank.

The 71-year-old white-haired physician was hailed as a "tireless worker in behalf of the citizens of West Bank, from the medical, political and civic standpoints."

The 50th anniversary was celebrated at Whiteside's Tavern in Gretna, where many of his friends joined in recalling days when Dr. Gelbke went his rounds in horse and buggy or wagon.

The doctor began his career immediately after his graduation from Tulane Medical School in May, 1900.

Dr. Gelbke was presented with a wallet-size gold plaque by Dr. J. S. Kopfler of Kenner as an expression of "love and esteem of your fellow practitioners on your 50th year of medical service." A leather wallet was also presented.

Dr. Charles R. Walters was guest speaker for the Central Medical Society at its meeting in Jackson, Mississippi, on April 4. The title of his paper was "Experiences with Thrombosis and Embolism on a General Surgical Practice."

REVISED EDITION OF MOTION PICTURE REVIEWS NOW AVAILABLE

The Committee on Medical Motion Pictures of the American Medical Association has completed the second revised edition of the booklet entitled "Reviews of Medical Motion Pictures." This booklet now contains 225 reviews of medical and health films reviewed in THE JOURNAL A.M.A. to January 1, 1950. Each film has been indexed according to subject matter. The purpose of these reviews is to provide a brief description and an evaluation of motion pictures which are available to the medical profession. Each film is reviewed by competent authorities and every effort has been made to publish frank, unbiased comments. Copies are available at a cost of 25 cents each from: Order Department, American Medical Association, 535 North Dearborn Street, Chicago, 10, Illinois.

WOMAN'S AUXILIARY OF THE LOUISIANA STATE MEDICAL SOCIETY

Doctor's Day was observed throughout the state with various forms of entertainment in honor of the physicians. The Calcasieu Auxiliary held a dance at the Majestic Hotel and the Ouachita Auxiliary gave a dinner at the Lotus Club. In Rapides Parish, Baptist Hospital and the Auxiliary jointly had a coffee party in the morning at the Hospital and presented each doctor with a red carnation boutonniere. Lafayette and Jefferson Davis Auxiliaries also had parties for the doctors.

Mrs. John S. Dunn, president of the State Auxiliary, made her annual visit to East Baton Rouge Auxiliary in March and was the principal speaker at their luncheon meeting.

Winners in the Hygeia essay contest, sponsored each year by the Calcasieu Auxiliary, were announced recently by Mrs. Walter Moss, chairman of the Hygeia Committee. The winners are Clara Viator and Curtis Duplechain from the Sulphur elementary school; Beverly Jane Graham and Wayne Fleming, West Lake school; John Sorrells, LaGrange school; Valda Gassee and Jimmy Rollins, DeQuincy school; Ann Mylius and Johnny Pecorino, Central school; and Donna McCurnin, Immaculate Conception. Seventh grade students of all Calcasieu Parish schools were invited to participate in the contest. The essay consisted of 300 words on a topic taken from Hygeia. Teachers selected the six best essays from each class and the Hygeia Committee chose the winners. Each winner was awarded a prize of $2.00.

At the April meeting of the Orleans Parish Auxiliary, Dr. Max M. Hattaway addressed the group on "What Organized Medicine is Doing to Improve Medical Care." The Orleans Parish Auxiliary donated $25.00 to the Cancer Fund and also contributed $25.00 to the State Auxiliary Commemoration Fund.

Registration of doctors who attended the 5th district Cancer Seminar was done by the Ouachita Parish Auxiliary.

At the March meeting of the Jefferson Davis Auxiliary, Mrs. Louis Shirley was the principal speaker. She chose as her topic "Jane Todd Crawford-Pioneer Heroine of Surgery."

A family has been "adopted" for one year by the Lafayette Auxiliary. The family which resides in Lafayette Parish consists of the parents and seven children who range in age from 1 year to 15 years. The father is a tenant farmer who is a surgical patient at Charity Hospital and who will need further surgery. The Auxiliary has agreed to send monthly contributions for food and to be responsible for the medical needs of the family. Individually, members of the Auxiliary will give canned goods, clothing, and shoes throughout the year. On holidays such as Easter, Thanksgiving and Christmas, the members will supply the family with appropriate gifts.

The 27th annual meeting of the Woman's Auxiliary to the A.M.A. will be held in San Francisco, June 26 to June 30. Headquarters will be at the Hotel Fairmont. A copy of the program may be obtained from Auxiliary's Office, 535 N. Dearborn Street, Chicago.

MRS. DANIEL M. KINGSLEY,
Chairman, Press and Publicity

BOOK REVIEWS

Clinical Laboratory Methods and Diagnosis: By R. B. H. Gradwohl, M. D., D. Sc., F. R. S. T. M. & H. (London) St. Louis, The C. V. Mosby Co., 1948, pp. 3148, Illus., Price $40. 3 vols.

This is the fourth edition of this text by Dr. Gradwohl, who on this occasion has had the collaboration of other investigators to produce such voluminous work that it has been divided into three volumes.

The first volume, entitled Urine, Blood, Gastric Analysis, Puncture Fluids, Feces, and Special Tests, covers comprehensively the greater number of commonly used technics involved with notations of the common sources of error. A fuller explanation of the principles involved in the above technic is recommended.

Volume II deals with Bacteriology, Serology, Post-mortem examinations, Tissues, Museum Toxicology, Crime, Basal Metabolism, and Electrocardiography. The section on Bacteriology is fairly comprehensive and offers an excellent fundamental treatise on this subject. The remaining portions of this volume as specified are more or less only outlines of the various procedures to follow.

Volume III, entitled Parasitology and Tropical Medicine, gives a brief history of the development of parasitology with a pertinent classification and definition of terms used. The section on malaria employs the Spanish classification and appears to be taken almost entirely from Kouri and Basneuvo's text of Parasitology and Tropical Medicine. The illustrations are very good, some being photographic, others drawings. The tables, particularly on malarial parasite development, are not in accord with the ideas of all investigators.

The whole text is an excellent encyclopedia of laboratory technic brought up to date.

E. H. LAWSON, M. D.

Modern Clinical Psychiatry: By Arthur P. Noyes, M. D., 3d ed. Philadelphia 5, W. B. Saunders Co., 1948. pp. 525. Price $6.00.

This is the third edition of this standard student-text since 1934. Well indexed, it conveniently provides pertinent bibliography at the end of each chapter. Dr. Noyes continues to remain on as solid ground as he can find in psychiatry, and although he presents the current concepts of psychopathology and dynamics, he does so with a critical scientific attitude and takes no sides. Dr. Noyes is still presenting schizophrenia in essentially Meyerian terms, i.e., "not as an autonomous disease entity, but as a type of reaction in certain personalities as a result of conflicts or complexes and a progressive difficulty in adaptation." He cites Kraepelin's and Bleuler's postulations of a structural-organic basis for the disorder but does not mention the more recent psychosocial-genetic concepts.

The "War Neuroses" are discussed with too much emphasis on the catastrophic combat reactions and too little on the vastly more common chronic anxiety reactions, hysterical reactions, and somatizations of everyday war service which fill most of the Veterans Hospitals' psychiatric beds today. Probably, too, many an ex-World War II medical officer familiar with the medical history of World War I will be astonished to learn that "Effort Syndrome" rarely occurred in World War II.

The chapter on psychopathic personality is quite elucidative for the non-psychiatrist in its presentation of the "Psychopathology of the Psychopath". Its breakdown of the Kraepelin and Kahn classifications of psychopathic personality, however, is as unnecessary as the classifications are superficial. Unfortunately, no mention is made of the Menninger classification of Pathological Personalities (Psychopaths) into schizoid personality, inadequate personality, etc., and this reviewer would consider the omission a serious defect, both because the terminology is the expression of a newer concept and is used by all Armed Forces medical departments and Veterans Administration hospitals and clinics, and because the problem of psychiatric nosology is an acute one, particularly to the record librarians of psychiatric hospitals. At the conclusion of the chapter Dr. Noyes mentions the Ganser Syndrome (nonsense syndrome) and cautions the reader that it should be distinguished from malingering. Under the psychoneuroses, he definitely terms it an hysterical reaction. It is wondered if many psychiatrists who have long dealt with criminals or medical pension-seekers still consider this syndrome "hysterical".

All in all, it is a good text for the student, the general practitioner, and the non-psychiatric specialist. The latter two, who so commonly express aversion for psychiatric jargon, will find this text reads easily for lack of it.

H. Tharp Posey, M. D.

Handbook of Medical Management: By Milton Chatton, A. B., M. D., Sheldon Margen, A. B., M. D., and Henry D. Brainerd, A. B., M. D. Univ. Medical Pub., 1949. 1st ed., 476 p. Price $3.00.

This handbook should prove of value where most recent and effective therapeutic measures for prompt and continued treatment are sought.

The data, though outlined, are easily grasped and are at once usable.

I. L. Robbins, M. D.

Occupational Marks; and Other Physical Signs: By Francesco Ronchese, M. D. Grune & Stratton, 1948. Illus. Pp. 181. Price $5.50.

This very interesting monograph should be enjoyed by any physician having the slightest avocation for deduction. Dr. Ronchese has organized and presented his material in a brief and concise manner so that the entire volume can be read in about one hour. More than half of the text consists of photographs which excellently illustrate the subject matter.

Dr. Ronchese demonstrates the many advantages of well-trained powers of observation. From a medical viewpoint, occupational marks that are immediately recognized as such often prevent an incorrect diagnosis or treatment; as for example psoriasis or keratoderma, in cases of callosities. In criminology, personal identification is frequently of inestimable value. Knowledge of occupational marks will also aid in establishing the social strata of the individual.

This monograph would seem to be especially valuable to all dermatologists who are called upon to diagnose and treat many of the stigmata of occupation or hobby. Shortly after reading this volume I had occasion to see a violinist's mark under the chin, a handwriting callus of the first phalanx of the middle finger in a clerical worker, and also, a callosity produced by scissors in a ribbon cutter. In every one of these cases treatment would be of no value unless the etiology were understood and in some cases corrective measures taken to prevent recurrence.

George Gaethe, M. D.

Human Nutrition: By V. H. Mottram, M. A. (Cant.) Baltimore, Williams & Wilkins Company, 1948. Pp. 267. Price $2.75.

This splendid little book on human nutrition, designed particularly for dietitians, is well planned and delightfully written. Facts are presented

clearly and interestingly in an easy somewhat conversational manner. It is refreshing when a scientific book retains accuracy and precision and yet has distinctive style and reflects the author's individuality and experience. The book deals largely with basic knowledge of the science of nutrition but includes many practical suggestions for applied dietetics. Among the subjects discussed are the role of calories, proteins, minerals, and vitamins in nutrition, digestion, absorption, and metabolism of foods, effects of cooking, processing, and storage on foods, and normal dietetics. This book should be useful to medical students as well as to dietitians, as a simple, accurate and up-to-date introduction to nutrition.

GRACE A. GOLDSMITH, M. D.

Pathology: Edited by W. A. D. Anderson, M. A., M. D., F. A. C. P., Professor of Pathology and Bacteriology, Marquette University School of Medicine, Milwaukee, Wisconsin. C. V. Mosby Co., 1948. Illus. Pp. 1453. Price $15.00.

This book has already established its position as an essential volume in the libraries of both pathologists and clinicians. Pathology, as have other branches of medicine, has expanded to such a degree, that the standard textbook is often inadequate. This book while following the pattern of standard texts has some 33 contributors who write with authority in their own special fields. It contains much more material than the average text and is an invaluable reference source. Of particular value are sections on special pathology, such as on skin by Arthur Allen, the organs of special sense by J. E. Ash, and bones and joints by Granville A. Bennett. Too often such important areas in the field of pathology are minimized in standard texts. Of necessity, in a work of this sort, there is some loss in homogeneity and continuity. Some sections are more thoroughly covered than others. On the whole, however, the balance is good. For sophomore students of pathology it is perhaps more detailed and voluminous than desirable in a beginner's text although as a supplementary text it is admirable. The bibliographies are excellent. Some of the omissions and minor errors inevitable in first editions undoubtedly will be rectified in later editions. We noted, for example, that there was almost no reference to sarcomas of the soft parts.

W. H. STERNBERG, M. D.

General Endocrinology: By Clarence Donnell Turner, Ph.D., Philadelphia. W. B. Saunders Co., 1948. Pp. 604. Price $6.75.

Experimental biology is presented as a fundamental aspect of biologic science, rather than as a medical specialty. Believing that the field of endocrinology can no longer be restricted to the study of a circumscribed set of ductless glands in the higher vertebrates, the author has broadened it to include comparable structures in lower and higher vertebrates. The regulation of growth processes in plants by compounds of a hormonic nature is also integrated.

Following the introductory chapters on the scope of endocrinology and biology of secretion, the glands of internal secretion, gastrointestinal principles, biology of sex and reproduction, hormones in pregnancy and lactation, and endocrine mechanisms in the invertebrates are presented from an experimental point of view.

Few clinicians have any conception of the varied and extensive research on this subject. In each chapter one finds revealing basic information as well as provocative experiments on the physiology of the endocrines.

The subject is regarded as the science of chemical coordination of the organism and this text is recommended to students preparing for medicine and allied branches as well as those interested in research on endocrinologic problems.

EUGENE H. COUNTISS, M. D.

Clinical Biochemistry: By Abraham Cantarow, M. D. and Max Trumper, Ph.D. Philadelphia, W. B. Saunders Company, 1949. Fourth Edition. Pp. 641. Price $8.00.

The latest edition of this excellent standard elementary text follows in order and emphasis the treatment of the subject in the earlier editions, but attempts to keep abreast of the rapid advances made in recent years. There has been some revision of the sections devoted to acid-base balance, carbohydrate, lipid and protein metabolism, thyroid function, adrenal functions, vitamins, and experimental diabetes, with the inclusion of much new material in diagrammatic and chart form, as well as significant additions to the well chosen bibliography that follows each sections of the book.

Newly added material includes consideration of chemical changes in shock, thymol turbidity and flocculation, fatty liver, lower nephron nephrosis, potassium in the treatment of diabetic coma, goitrogenic agents, and new methods of studying adrenocortical function. The text has been kept to the same length as the previous edition, and is superior to it in physical make-up.

A. O. KASTLER, Ph.D.

PUBLICATIONS RECEIVED

University Medical Publishers, Palo Alto, Calif.: Handbook of Obstetrics and Diagnostic Gynecology, by Leo Doyle, M. S., M. D.

Charles C. Thomas, Publisher, Springfield, Ill.: Treatment in Psychiatry, by Oskar Diethelm, M.

D. (Second Edition); The Nose; An Experimental Study of Reactions Within the Nose in Human Subjects During Varying Life Experiences, by Thomas H. Holmes, M. D., Helen Goodell, B. S., Stewart Wolf, M. D., and Harold G. Wolff, M. D.; Chemical Developments in Thyroidology, by William T. Salter, M. D.; B. C. G. Vaccination in Theory and Practice, by K. Neville Irvine, D. M., M. A.

Grune & Stratton, Inc., New York: Progress in Clinical Endocrinology, edited by Samuel Soskin, M. D. (Second Edition).

Northland Press, Saint Paul: SAW-GE-MAH (Medicine Man), by Louis J. Gariepy, M. D.